UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF NORTH CAROLINA
ASHEVILLE DIVISION

STATE OF NORTH CAROLINA ex rel. Roy Cooper, Attorney General, Plaintiff,	)	No. 1:06-CV-20
vs.	)	**VOLUME 10A**
TENNESSEE VALLEY AUTHORITY, Defendant.	)	[Page 2330-2466]

TRANSCRIPT OF TRIAL PROCEEDINGS
BEFORE THE HONORABLE LACY H. THORNBURG
UNITED STATES DISTRICT COURT JUDGE
JULY 25, 2008

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PLAINTIFF'S EXHIBITS

NUMBER	ADMITTED
493	2466

DEFENDANT'S EXHIBITS:

NUMBER	ADMITTED
342-344	2339
345,346	2420
364	2444
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P R O C E E D I N G S

THE COURT: All right, Mr. Lancaster. You may call your next witness.

MR. LANCASTER: Thank you, Your Honor.

Defendant Tennessee Valley Authority calls Dr. Suresh Moolgavkar.

THE COURT: All right.

SURESH MOOLGAVKAR,

being duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

MR. LANCASTER: I would ask that Dr. Moolgavkar take exhibit book 14 from the shelf next to the witness stand, and the Court may wish to have reference to defendant's book 14.

THE COURT: All right, sir.

BY MR. LANCASTER:

Q. Dr. Moolgavkar, could you please state your full name for the record?

A. My name is Suresh Moolgavkar. Would you like me to spell it?

Q. Yes, sir.

A. First name is Suresh, S, as in Sam, u-r-e-s-h. The last name is Moolgavkar, M as in Mary, O-O, L as in Lion, G as in George, A as in apple, V as in Victor, K as in King, A as in apple, R.

Q. Dr. Moolgavkar, where do you live?

A. I live in the Seattle area, in a suburb of Seattle called Bellevue, Washington.

Q. You have been retained by Tennessee Valley Authority as an expert in this lawsuit?

A. Yes, I have.

Q. And you have authored three reports?

A. Yes, I have.

Q. And are Defendant's Exhibits 342, 343 and 344 copies of those reports?

A. May I take a look at those?

Q. Yes, sir.

A. Yes, they are.

Q. And you are an epidemiologist; is that correct?

A. That is correct.

Q. How long have you worked in the field of epidemiology?

A. For more than 30 years now.

Q. What is your educational background?

A. I got a medical degree from the University of Bombay, completed one year of residency there, and then came to the Johns Hopkins University Medical School as a post-doctorate fellow in pharmacology and biophysics.

While at Johns Hopkins, I obtained a Ph.D. in mathematics and got post-doctorate training in epidemiology and biostatistics at the University of Washington.

Q. Where are you currently employed?

A. I'm currently employed by a consulting company, an international consulting company called Exponent.

Q. And until recently, you were employed in an academic setting; is that correct?

A. That is correct. In fact, technically, I'm still a member of the faculty of the University of Washington and of the Fred Hutchinson Cancer Research Center in Seattle. However, I will be retiring from those positions and taking emeritus status as of August 1st of this year.

Q. That's next week.

A. Next week, yes, sir.

Q. And you taught at other places besides the University of Washington; isn't that right?

A. Yes. I have taught at the Johns Hopkins University, at the University of Pennsylvania, at Indiana University in Bloomington, and, then, finally at the University of Washington.

Q. And have you been at a visiting scientist at any place?

A. Yes. I've held several visiting scientist positions.

I've been a visiting scientist at the International Agency for Research on Cancer in Léon, France. That is the cancer research organization of the World Health Organization. I have been a visiting scientist at the Radiation Effects Research Foundation in Hiroshima, which was

set up after the Second World War to investigate the effects of ionizing radiation on the populations of Hiroshima and Nagasaki. I've been a visiting scientist at the German Cancer Research Organization in Heidelberg. And I have also spent brief periods of time at the Canadian -- equivalent of the Canadian EPA.

Q. And you have published a number of articles and papers in your field?

A. Yes. I have over 150 articles in the field of epidemiology, biostatistics and risk analysis.

Q. I would like to read briefly from an article that was attached to the expert report of Dr. Peden which has already been admitted in this case. It's an article published by Dr. Pope and Dr. Dockery in 2006.

It indicates: "Beyond simply recognizing gaps in knowledge, there remains a need for a healthy skepticism regarding what we may think we know about the health effects of particulate matter exposure. There are important scientific issues that are not fully resolved. For example, Moolgavkar argues that potential confounding, measurement error, model building and selection, and related issues remain concerns, especially when the estimated risks are small."

Are you that Moolgavkar?

A. I believe I am.

Q. So your work has been cited in the article relied upon by Dr. Peden?

A. Yes.

Q. And in addition to publishing articles, have you had editorial responsibilities in the field of epidemiology and related disciplines?

A. Yes. I was one of the editors of a journal called *Genetic Epidemiology* in the 1980s. I edited several books and monographs in the field of epidemiology and biostatistics. I'm currently one of the editors of *Risk Analysis: An International Journal*, and also on the editorial board of *Inhalation Toxicology* and of the web-based journal called *Biology Direct*.

Q. Are you a member of any organizations of epidemiologists?

A. Yes, I'm an elected member of the International Epidemiology Society.

Q. Have you sat on any review panels as a consultant on epidemiology?

A. Yes. I've been on several National Institutes of Health review panels as an expert reviewer for grants and as a site visitor at various cancer centers site visits.

I have served as a consultant to the California Air Resources Board. I've served as an consultant to the USEPA, to the Department of Energy, to the World Health

Organization, to the -- I'm also one of the scientific advisers to the European Union effort to estimate the risk from low doses of ionizing radiation. In fact, I chaired that panel.

I've also been a consultant to Health and Welfare Canada.

Q. And is Health and Welfare Canada simply a Canadian EPA?

A. Yes, it is.

Q. In your service to these organizations, have you had experience specific to evaluating the health risks of air pollution, including particulate matter?

A. Yes. In fact, that is my main role as an adviser to the California Air Resources Board.

And I forgot to mention that I was also on an HEI -- that is Health Effects Institute -- advisory panel that was involved in oversight of reanalysis of the Harvard Six Cities and the ACS-2 studies.

Q. And ACS is short for American Cancer Society?

A. Yes. That's one of the largest cohort studies that has been done to look at the risk of exposure to fine PM.

Q. And you've testified as an expert on epidemiology before?

A. Yes, I have.

Q. In court?

A. Yes.

Q. How about other places?

A. I'm not sure. What do you mean by testify?

Q. Have you testified before EPA's Clean Air scientific Advisory Committee?

A. Yes. Yes, I have.

Q. In this case, have we asked you to address the question about whether the opinions expressed by plaintiff's experts are supported by the current available epidemiological literature on the association between air pollution and human health?

A. Yes.

Q. And you have formed conclusions on that matter?

A. Yes, I have.

Q. And are they described fully in your written reports?

A. Yes.

MR. LANCASTER: Your Honor, we tender Dr. Moolgavkar as an expert in the field of epidemiology, specifically including air pollution epidemiology.

MR. GULICK: Your Honor, as I had told Mr. Lancaster before this began, we'd stipulate to Dr. Moolgavkar's expertise as he has just stated.

THE COURT: All right. Let the record reflect the stipulation, and show that the Court holds Dr. Moolgavkar to be an expert in the field as articulated by TVA counsel.

MR. LANCASTER: Thank you.

Thank you, Mr. Gulick.

I would also move the admission of Defendant's Exhibits 342, 343 and 344, which are Dr. Moolgavkar's expert reports.

MR. GULICK: With respect to those, Your Honor, for the record, understanding how Your Honor has ruled on these, we had filed a motion in limine with respect to these, so we would ask that they be excluded in accordance with that motion in limine.

But that's for the record. I understand Your Honor has taken these into evidence already.

THE COURT: Yes. Show the objection overruled.

MR. LANCASTER: Thank you.

(Defendant's Exhibit No. 342, 343 and 344 received in evidence.)

BY MR. LANCASTER:

Q. Now, Dr. Moolgavkar, I'll have to confess that when I began working on this case I could barely pronounce the word "epidemiology," much less know what it was.

Would you explain to the Court the basics of what the science of epidemiology is.

A. Yes. I'll try to do it as briefly and succinctly as I can.

Epidemiology is basically the study of diseases in populations, but mostly, of course, interested in human

population, so let me restrict my attention to epidemiology as the study of diseases in human populations. It is usually defined as the study of the distribution and determinants of diseases in populations.

Epidemiology is largely an observational discipline. This means that the kind of randomized trials or randomized experiments that can be done in the laboratory cannot be done in epidemiology because, obviously, it would be unethical to randomize human beings to one exposure or another. So what you see is basically what you get. So that is why it is an observational discipline.

I do want to make clear that there are occasions when clinical trials or the equivalent of clinical trials can be done in epidemiology, and that is important to keep in mind because that is the only study designed which can eliminate the problem of what is called confounding, which I will explain in just a minute.

And so let me explain to you, Your Honor, what I mean by confounding. Since epidemiology is an observational discipline, and, as I said, you cannot randomly assign human beings to one exposure or another. So if one wants to study air pollution and the effect of air pollution on human health, then, of course, the most obvious design would be to take groups of human beings and assign one of them randomly, one group randomly, to no exposure and another group to

exposure, obviously, we cannot do such a thing. So we have to use what we can observe in the population.

Now, as a simple example, we know that levels of air pollution are highly correlated with weather. So on hot, humid days, for example, when the air is stagnant, air pollution is also high, and if you observe increased mortality on the next day after an episode of high air pollution but also an episode of very high heat and humidity, it is difficult to verify the reason for that mortality. Is it due to air pollution or is it due to high heat and humidity? So that is a concrete example of a confounder.

In this case, weather is a confounder of the air pollution effects, and unless you can adequately control for weather, you cannot conclude that air pollution had anything to do with mortality on the next day.

So that is one of the most important jobs of the proper epidemiological analysis, to control for confounding such as that.

So to give a general definition, a confounder is a kind of hidden variable in the investigation of epidemiological studies, which, if not properly and adequately controlled for, can lead to biased conclusions about an association between the exposure of interest and the disease.

So that is one big problem we run into because it's an observational discipline.

The other big problem, and that is particularly to air pollution epidemiological -- it occurs in other types of epidemiological studies also, but it's very important in air pollution epidemiology, and that is the proper measurement of exposure. By that I mean, if you want to look -- study the effect of cigarette smoking on lung cancer, you can go to the subjects and ask them to tell you what their smoking habits are: How many cigarettes did you smoke? When did you start smoking? When did you quit smoking? So you can get information on the individual level. However, in air pollution epidemiology, we cannot measure what individuals are exposed to. All we have are central monitors where readings are taken, and then we assume that everybody who is in the proximity of that monitor would be exposed to the same level of air pollution; although, clearly, individual exposure will depend upon the habits of the individual, how much the individual is outside the home, how much the individual -- how much time the individual spends inside in an air conditioned home or an air conditioned office, and so on. So the circumstances of exposure for each individual might be quite different, and, yet, we simply assign an exposure that is measured at a central monitor.

So these are the two big problems for epidemiology, and specifically for air pollution epidemiology: Confounding and mismeasured exposure, or exposure that is not well known.

And, therefore, air pollution epidemiology is particularly difficult to study.

The problem is made even more difficult because the risks that we are trying to eke out in air pollution epidemiology are so small. The risks of smoking-associated lung cancer are pretty large, and so they are easy to separate -- it's easy to separate the signal from the noise. But in air pollution epidemiology, the risks are pretty small, and so it becomes a very difficult task to eke out the signal and separate it from the noise. And for that reason, a number of very sophisticated statistical techniques have been devised for the study of air pollution epidemiology.

Now, I need to introduce one more concept, Your Honor, and that is the concept of how you measure the association between an exposure of interest and a disease. And, generally, the measure that we use in epidemiology is called a relative risk. And the relative risk very simply defined is defined as the ratio of the risk in the exposed population divided by the risk in the unexposed population. So you're simply looking at the proportionate increase or decrease in risk associated with the exposure. So if the relative risk is greater than one, then you say that there is a positive association between the risk factor and the disease. If the relative risk is less than one, you say that there is a negative association between the risk factor and the disease.

But having measured the relative risk, you can only conclude that there might be an association between the risk factor and the disease.

There is one more step that needs to be taken, and that step is to look at statistical significance of that relative risk.

So the question we are really asking here is that, when you do an epidemiological study and you come up with some estimate of relative risk, that is, some estimate between the association of the risk factor and the disease, then the question becomes, could that just be a fluke? Could that just have a good by-chance? And the statistical method that is used to test that is the idea of statistical significance.

So if the association between the risk factor and the disease is deemed to be statistically significant, then what you are saying is that this association has probably not occurred by chance; it's a real finding.

And to give an analogy which is I think particularly timely in this election season, when you take polls and you come up with numbers like 41 percent and 44 percent and say that, well, the difference is within the margin of error, what you're really saying is that the difference is not statistically significant and 41 is not really different from 44 percent.

So that is the idea. What is the probability that this

finding in an epidemiological study is a good by-chance, and you use statistical significance to get a measure on that question.

Now, having said that, even if you find a statistically significant result in one epidemiological study, because of the problems that I've described, mainly those of confounding and error in measurement of exposure, that is not generally sufficient to compute that the exposure might be related to the disease in a causal way. In order to do that, you need quite a bit more. You need to do many epidemiological studies in different populations and under different circumstances, and if the results of all these epidemiological studies are pretty consistent and if the associations are pretty strong, then we might decide that the association is causal. However, if the associations are not really strong, if they're weak, that is, if the risks are very small, then not only do you need a number of consistent epidemiological studies, but we also need to have some biological basis for thinking that the association might be causing it. So some sort of a biological plausibility needs also to be established before saying that the association is causal.

So I think that, very briefly, Your Honor, is a tutorial on epidemiology and the pitfalls of doing observational studies rather than randomize the clinical trials.

And I would say that this is not just an academic concern. This is a real concern. And the issue of confounding in observational studies has led to some very serious mistakes in the past. And one of the examples of where a very serious mistake was made, because -- in good faith, because of the issue of confounding to not be addressed adequately, is that the issue of hormone replacement therapy for postmenopausal women.

In the 1990s, based on a whole slew of observational studies, epidemiologists were pretty sure that administration of hormone replacement therapy to postmenopausal women was really good for them because it prevented a whole host of conditions, including bone fractures and, quite importantly, cardiovascular disease. And it all seemed to make biological sense because women have a lower risk of cardiovascular disease than men, and it was hypothesized that it was because of their hormone profiles. And so there was a whole host of biological reasons also to believe these observational epidemiology studies that giving a postmenopausal -- giving of hormone replacement therapy to postmenopausal women would protect them against heart disease.

And, in fact, two professors at Harvard, Graham Colditz and Mile Stanford, wrote an editorial and actually did an analysis of all the population and epidemiology studies and concluded in the 1990s that, in fact, it would be unethical

not to give postmenopausal women hormone replacement therapy.

Now, in the early 2000s, at the behest of the NIH, a very large clinical study, clinical trial, was undertaken to look at many, many different issues of women's health, and one of the things that they undertook was a proper randomized clinical trial of hormone replacement therapy in postmenopausal women, and at the end of that trial, lo and behold, what did the investigators discover? They discovered that hormone replacement therapy was not good for women. Not only did it not provide any protection from heart disease, in fact, it increased to some extent the risk of heart disease. So all these observational epidemiology studies had been wrong for these many years, and the clinical trial was undertaken and the cart was upset.

And what is the reason for this? The reason that has been put forward is confounding by socioeconomic factors, or inadequate control of confounding by socioeconomic factors. Because well-to-do women have access to physicians, they are more likely to take hormone replacement therapy than poor women. The have access to physicians; they eat a better diet in general; they exercise more than poor women do. In general, they lead a healthier lifestyle. And because of that, it turned out that the conclusions of the observational studies was that hormone replacement therapy was good. Actually, the conclusion should have been that women in the

upper socioeconomic status had better heart health than poor women.

So here is a case of confounding by socioeconomic status which really led to findings that were quite biased and it led to a standard of practice for many years in this country that was probably wrong.

Q. So the hormone replacement therapy example is one where the failure to properly address what you called confounding led to some unfortunate results?

MR. GULICK: Object to leading.

THE COURT: Overruled.

THE WITNESS: Yes. That is one example. And I think it's particularly relevant to the air pollution example, because confounding by socioeconomic status is particularly relevant to air pollution because, of course, it's the poor people at the lower end of the socioeconomic status that are also exposed to the greatest amount of air pollution. And I will come back to this when we talk about the American Cancer Society studies.

BY MR. LANCASTER:

Q. Okay. Thank you.

And this confounding problem is common to all air pollution -- excuse me -- I mean all epidemiological studies, but particularly so for air pollution studies.

A. Yes. It is particularly relevant for air pollution

studies because, first of all, the risks that you're trying to detect are extremely small. So any kind of small confounding could bias the risks. And, secondly, the confounders are also very complex.

So, for example, if you want to look at the risks associated with fine particulate matter, then you have to consider confounding by a whole host of other pollutants. I mean, you have to consider sulfur dioxide, nitrogen dioxide, ozone, and, in reality, you should also be considering all the pollutants in the environment that are not even routinely measured upon which we have no information; and then, again, this confounding by weather, as I have already said.

Lifestyle factors could also confound the association between air pollution and health. So, for example, cigarette smoking habits need to be taken account of in certain types of studies.

Q. Okay. Dr. Moolgavkar, are you aware that Dr. Levy has testified to a calculation of the number of premature mortalities that he contends will be avoided if TVA's projected emissions are reduced and, as a result, the PM2.5 levels in North Carolina are reduced?

A. Yes, indeed, I'm familiar with those.

Q. What is your opinion about Dr. Levy's calculation?

A. Well, I don't believe that those calculations are based on sound science and I don't think they're defensible. But

before I get into the technical epidemiological and statistical reasons, I think that I would like to make a sort of general statement, and that is statistics is a method or a tool for looking at complex relationships in databases, but this statistical methodology -- but I would say statistics does not trump common sense. If statistical methodology yields answers that run counter to common sense, then I think it's incumbent upon you to take another look at the statistical analysis that you've done to see whether the numbers you get make any sense of.

Now, if you look at the calculations done by Dr. Levy -- let me say this. First of all, if you believe that air pollution is associated in a causal fashion with health effects on human populations, then what you would expect is that on or after high-pollution days you would expect a certain number of people to get sick, either respiratory disease or, what is now generally believed in the air pollution literature, cardiovascular disease. So you would expect a certain number of people to get sick and be admitted to the hospital and then, for a certain smaller fraction of that number to die because of exposure to air pollution.

So when you look at calculations of the number of lives saved and the number of hospital emissions avoided, what you would expect to see is a large number of hospital admissions avoided and a smaller number of deaths avoided.

However, if you look at Dr. Levy's calculations here --

Q. I don't mean to interrupt you, but Ms. Shea has been good enough to put Plaintiff's Exhibit 231 on the screen, which is Dr. Levy's summary of his calculations for North Carolina.

A. Right. So if you -- Your Honor, if you look at premature mortality, Dr. Levy calculates that 99 cases would be avoided. So he's basically saying that 99 cases per year are caused by exposure to emissions from TVA. But if you look at the total number of hospital admissions that would be avoided, that's only 60. That is much less than the number of deaths that would be avoided. And so what Dr. Levy's calculations imply is that a lot of people are simply falling dead without ever being admitted into hospital. And this is something that common sense tells me is highly unlikely. It is not a result that I can believe.

There is another thing here that also sticks out. In the air pollution literature, the focus currently is on cardiovascular disease, not on respiratory disease. Respiratory disease has been played down. And most of the deaths that are talked about in the air pollution literature are due to cardiovascular disease, not due to respiratory disease. And yet, in this table, Dr. Levy has twice as many respiratory hospital admissions as cardiovascular hospital admissions.

So, to me, these numbers, just from the common sense point of view, really are difficult to swallow. I cannot accept these numbers for that reason.

Well, then to get into the more epidemiological and statistical reasons, I think we have to look at the generally inconsistent epidemiology literature. Now, there are two classes of study that have been looked at by Dr. Levy, and the first class of studies is the so-called long-term studies that look at the long-term effects of air pollution on mortality, and the other are the so-called time-series or short-term studies.

Q. Go right ahead, sir.

A. So let me talk about the long-term studies first because that's where Dr. Levy's calculation of premature mortality comes from, so that is the most important part for PM. Premature mortality associated with fine PM comes from the long-term studies.

Now, long-term studies are difficult to do, expensive, so there are few of them. Half a dozen long-term studies have been done. Many of them have shown no association between fine PM and mortality. Examples are studies done by Enstrom in California, the studies done by Lipfert on the Veterans score, and also, the second half of the study done by Laden on the Six Cities score.

These studies show no association between fine PM and

mortality.

Q. Let me stop you right there briefly.

Are you saying that the Harvard Six Cities Study that Dr. Levy cited actually shows no association?

A. I believe that the extension of the Six Cities Study published by Laden, et al, one interpretation of that study is that during the second period over which they looked at the data, there was no association between fine PM pollution and mortality.

Q. Okay. Go right ahead, sir.

A. So these studies, particularly, the Enstrom and Lipfert studies, Dr. Levy completely ignores. So let's set those aside. But let's look more critically and carefully at the families of studies that he does consider.

He considers some of the studies done -- some of the studies based on the Six Cities data, and he also looks -- very important part -- at the coefficient. I should say that the coefficient that he uses to derive his estimates of mortality comes from the, basically, from the American Cancer Society study. So let's look at these two families of the Six Cities Study and the American Cancer Society studies.

Now, because of the number of criticisms addressed in the original Six Cities Study gathered up in 1993, the study was reanalyzed under the auspices and funding from the Health Effects Institute in the year 2000, and the reanalysis was

conducted by Dr. Krewski of the University of Ottawa. And here is what they found.

What they found was that not only was fine PM associated with mortality, but all the other gases were associated with mortality as well in about the same way, with the single exception of ozone. So in the Six Cities long-term study, ozone was in no way associated with mortality, but the other gases were, and because there were only six cities in the study, confounding by the gases would not be addressed in this study. So Krewski, et al, say that it's impossible to tell from the Six Cities Study whether it's fine PM or one of the other gases that is associated with mortality. Could be any one of them. You cannot test that hypotheses in this data set.

So the Six Cities Study really does not provide very strong evidence that it's fine PM that is associated with mortality. And, in fact, let me reiterate that, for ozone, which is the other pollutant of interest in this case, the Six Cities Study found absolutely no association between levels of ozone and mortality.

Now let's go to the second family of studies, that based on the American Cancer Society cohort, and that is a much more interesting study because it is much larger. It involved almost half a million individuals. It was first published by Arden Pope and others in 1995. It came under

for the same kind of criticism as did the Six Cities Study. Namely, they did not take confounders into account. Even though they could have looked at the gases at the time, they did not. They simply looked at fine PM and nothing else in their analysis. So this study was also reanalyzed by Krewski, et al.

And by the way, as I said earlier, I was on the scientific advisory panel, external advisory panel for this reanalysis of both the Six Cities and the American Cancer Society studies.

So when the American Cancer Society study was reanalyzed by Krewski, et al, here were the main findings. First, it was fine particulate matter, or specifically sulfate, that was most strongly associated with mortality. It was, indeed, one of the gases. And the gas that was associated with mortality was sulfur dioxide. So when you considered sulfur dioxide in an analysis simultaneously with fine particulate matter, sulfur dioxide was the pollutant that was significantly associated with mortality, and the fine particle signal basically vanished, became highly insignificant.

Now, it is generally agreed, there is a consensus that biologically there is no basis to suspect that sulfur dioxide could be causing death. Therefore, the association of sulfur dioxide with mortality remains unexplained at this time. We

don't know what the reason is. But we do know that when you consider sulfur dioxide, fine PM is no longer associated with mortality in the ACS, the American Cancer Society, study. That was one finding.

The second finding of Krewski, et al was that when you looked at just fine PM, just the fine PM and mortality, without looking at the confounders, which is what Dr. Levy does, that effect of fine PM or the association of fine PM with mortality was restricted to people only with a high school education or less, in other words, to people in the lower socioeconomic strata. There is no reason to believe that air pollution would differentially kill off people in the lower socioeconomic strata. And that is another flag that there are certain socioeconomic factors that have not been adequately adjusted in the study.

The third fact. In a statistical analysis, it is important to make allowance for the fact that cities that are close together have highly correlated mortality rates. So, for example, if you look at Minneapolis and St. Paul, mortality rates are highly correlated in those two cities for reasons that we do not completely understand. The same thing would be true of Miami/Ft. Lauderdale, for example. Mortality rates would be correlated for reasons that we do not completely understand. And it's important to take that kind of correlation into account. And when Krewski, et al,

did an analysis taking this kind of spatial correlation into account, they found that fine particles and sulfates were no longer significantly associated with mortality.

For a fact, when you look at some of the analyses done by Krewski, he found, for example, that in the Six Cities Study, ozone and nitrogen dioxide -- not Six Cities. Sorry. When you look ACS study -- I'm still talking about the American Cancer Society -- both ozone and nitrogen dioxide were negatively associated with mortality and they were statistically significant. So what this data were saying, that ozone and nitrogen dioxide have a protective effect on human health, which, of course, is biologically highly implausible and one cannot accept that finding, of course, at face value. But in view of that finding, it is difficult to take any of the other quantitative estimates given in that study seriously.

So if you get findings of this type that are simply counter to biology, how can you take any of the other conclusions of the study seriously, at least in a quantitative sense?

Now, in further analyses of the American Cancer Society study, there was another paper that appeared in 2002, in JAMA, *Journal of the American Medical Association,* again by Arden Pope, et al, the person who wrote the first paper in 1995, and in that paper he reports in a figure that carbon

monoxide is also protective of cardiovascular mortality, that is, it is negatively associated with cardiovascular mortality, and that is statisticallly significant. Again, that is a finding that is counter to all of biology, particularly in view of the fact that carbon monoxide is a cardiac toxin. And so that finding is unbelievable. But I think it is wrong to ignore that finding and simply to accept the finding on fine PM from that study.

So, once again, I think the quantitation of the association between fine PM and the mortality in that paper cannot be taken seriously because there are other results. The paper is not entirely consistent with respect to the biology.

Then in another paper published in 2004, in *Circulation*, the journal *Circulation*, Pope, et al, looked at both respiratory disease and cardiovascular disease and they found a specifically significant negative association between fine PM and respiratory mortality. And that again does not make biological sense.

So if the papers are not consistent with respect to biology, it is difficult to take the precise quantitation that they offer for the association between fine PM and mortality very seriously.

So, for this whole host of reasons, I feel that the precise coefficient that Dr. Levy has used and which is

derived mainly from consideration of the American Cancer Society studies, that the precise coefficient that he has used to derive the number of deaths in North Carolina, or anywhere else in the domain in which he has calculated these deaths, is just totally unjustified. Cannot be done.

Q. Thank you, Dr. Moolgavkar.

And are there, in addition to the two major families of long-term studies about which you've described some of the issues, those being the Harvard Six Cities Study and the American Cancer, ACS, study that was reanalyzed by Dr. Krewski, K-r-e-w-s-k-i, are there also short-term studies, or what you call time-series studies, that look at pollution even in North Carolina?

A. Yes, there are. In fact, I think the single most important short-term, or time-series, studies of air pollution and mortality are the so-called NMMAPS studies, National Mortality and Morbidity and Air Pollution Study.

Q. So that's N-M-M-A-P-S?

A. Yes, N-M-M-A-P-S.

These studies were funded by the Health Effects Institute, the same institute that funded the Krewski reanalysis, and they were conducted by investigators at Johns Hopkins University and they remain the most comprehensive short-term studies of the association between air pollution and mortality undertaken in the United States. And here

again, the fundamental fact is that they looked at 90 cities across the United States, including some in North Carolina. So that is extremely relevant to the North Carolina situation. And when they looked at particulate matter, they found no association between particulate matter and mortality in three -- in the three North Carolina cities they looked at. They looked at three North Carolina cities, Raleigh-Durham, Charlotte and Greensboro, and they found no association with particulate matter and mortality in those three cities.

They also looked at two cities with respect to ozone. That is Raleigh-Durham and Charlotte. For some reason they didn't look at Greensboro for ozone, probably because ozone readings are not available in that city. But even also for ozone, as for particulate matter, there was no association between ozone and mortality in these two North Carolina cities.

Now, let me state one sort of overarching kind of observation here. Dr. Levy bases his calculations of mortality from the exposure to fine PM on the long-term studies, basically, from the coefficients derived from the American Cancer Society study, but these long-term studies found no association between ozone and mortality, and, therefore, Dr. Levy does not -- he ignores these studies and he goes to the short-term studies to assess the impact of

ozone on mortality. And he does not look at the specific results in North Carolina, but he looks at some overall national result from the NMMAPS studies and does his calculation in that way. And so he is using the long-term studies for fine PM. He's using the short-term, time-series studies for ozone because the long-term studies don't show any association with ozone.

So the question I have is how can air pollution in the short term increase mortality from ozone and this does not show up in the long-term statistics? How can you have short term associations between ozone and mortality without that showing up in the long-term studies?

So I think there is some sort of a logical disconnect here in the way that Dr. Levy has done his calculations.

Q. There has also been testimony, Dr. Moolgavkar, that the particulate matter component at issue here is sulfate. What is -- and you've made some reference to sulfate in your testimony so far. Could you briefly summarize the status of the epidemiological literature of the relation between sulfate and mortality?

A. Yes. In fact, I think one of the assumptions made by Dr. Levy in all his calculations is that all fine PM is, if we accept -- if we accept -- that fine PM is causing the association with mortality. And I've given you a whole host of reasons now to at least question that conclusion for the

current ambient levels of fine PM.

Another assumption made by Dr. Levy is that all particles are equal in their toxicity. And there is a fair bit of literature, not a huge amount, but there is a fair bit of literature indicating that all fine particles are not equal in their toxicity. There are time-series studies done in Atlanta by Lam, et al. There is the new Lipfert study that also concludes that components of fine particles may have different toxicities. There are other Canadian studies that by Burnett, et al, that also show components of fine PM mix are important. And then, finally, there are studies by Dr. Levy's colleagues. Dr. Laden and others published a study in 2000. I have a number of concerns about the way that study was done and some of its conclusions. However, if one accepts their conclusions, they show that particles from vehicular sources are three times -- at least three times as toxic as sulfates emitted from power plants.

In a later study by Maynard, et al, that appeared in 2007 -- I believe these are also colleagues of Dr. Levy -- they also found that, in fact, when you look at particles from power plants and compare them and look at them jointly with particles from vehicular emissions, it turns out that the particles from the power plants are not significantly associated with mortality, whereas the particles from vehicular emissions are.

So, again, these studies point to the fact, first of all, that not all particles are equal in their toxicity; and, secondly, even if you look at the few studies that have tried to parse out the source of particles, it is the vehicular emissions that appear to be a lot more toxic.

Q. What does the inconsistency in the literature you've been describing mean in terms of reaching causal conclusions and quantitative estimates?

A. Here is what I believe based on all the literature that I have looked at. First of all, I think the literature is inconsistent, and for the same data set, depending upon which model you use for the data, and how you control confounding by weather and co-pollutants and so on, can make a huge difference to the results of the analysis. So the data are pretty sensitive to the method of analysis. So you don't get consistent results.

So, based on that, my personal belief is could air pollution and fine particulate matter, in particular, be associated with health effects at the current low levels in the United States? Yes, it could. You can't rule it out. But can you definitively assert that there is a causal connection? I don't believe you can. And neither can you come up with any precise estimate of the dose response coefficient that would allow you to estimate the number of deaths avoided -- that would be avoided if further controls

were imposed on TVA.

Q. Thank you, sir.

Finally, I want you to address one last topic.

Dr. Levy testified that he provided no quantitative uncertainty bounds or sensitivity calculations for his premature mortality calculations, although it would be very important to do so to publish the work in a peer review journal.

Do you have any opinion about the validity of his failure to do that?

A. Well, I think that in any exercise of this type, one has to provide uncertainty estimates, because his estimates are based on a whole chain of models that he has used. He has estimates of emissions, he has models for air dispersion, models that tell him what the exposure would be in any given area, then he has a model for the dose response that he has derived basically from the American Cancer Society study. Each one of those steps in the process of providing the estimates of the number of deaths averted, each one of those steps is subject to uncertainty and to error, and that uncertainty is extrapolated down the chain.

What Dr. Levy should have done in this case is applied a method called Monte Carlo simulation in order to come up with some uncertainty estimates on the numbers that he provided. And, in fact, in previous papers with Dr. Spengler, Dr. Levy

has done just that. He has provided uncertainties.

Q. In his published papers?

A. In his published papers, yes.

Q. In summary, Dr. Moolgavkar, is it your conclusion that the epidemiological studies cited by plaintiff's experts cannot be used to derive scientifically with reliable estimates of alleged health impacts from the TVA power plants under the circumstances of this case?

A. I do not believe, in my expert opinion, that these can be used the way they've been used by Drs. Levy and Spengler.

MR. LANCASTER: Thank you.

Your Honor, no further questions.

THE COURT: Mr. Gulick?

CROSS EXAMINATION

BY MR. GULICK:

Q. Good morning, Dr. Moolgavkar.

A. Good morning.

Q. First, Dr. Moolgavkar, you indicated that you had a medical degree from the University of Bombay, but you do not actually practice medicine, do you?

A. No, I don't currently practice medicine, and I haven't for many years. I did take my licensing exams in the United States but never did practice medicine.

Q. And you got your medical degree in 1965.

A. In 1965.

Q. And then you practiced during your residency?
A. Yes, sir.
Q. And that was the last time you practiced medicine?
A. Correct.
Q. And, so in the last 40 years, you've not practiced medicine?
A. That is correct.
Q. And I believe you indicated that you are not a toxicologist; is that correct?
A. I have done some work in toxicology. I've published a few papers, but I'm not a toxicologist.
Q. And I believe you testified that you do not do chamber studies; is that correct?
A. I do not do chamber studies.
Q. And it's also true, is it not, that you have never conducted a long-term study in the type of the American Cancer study or the Six Cities Study?
A. There are probably a half a dozen individuals in the world that are involved with an air pollution study of that type.
Q. So the answer is no?
A. No. The answer is no, I have not been directly involved in a study of that type.
Q. But you yourself have performed a few time-series studies?

A. I have done numerous time-series studies, yes.

Q. Doctor, I believe you indicated in your testimony that a considerable portion of your career was devoted to studying, in an epidemiological manner, cancer. Is that correct?

A. Well, the main focus of my career over the last 30 years has been cancer epidemiology, but for about the last 10 or 12 years, I've also done quite a bit in air pollution epidemiology.

Q. I understand. And if you would turn to your CV, Dr. Moolgavkar, which I believe is found at the end of your first report, which I believe is Defendant's Exhibit 342.

A. Yes, I have it.

Q. And it's an attachment to your first report, is that correct?

A. That's correct.

Q. I'd like to direct your attention to page No. 7 of your CV.

Have you found that?

A. Yes.

Q. And I want to direct your attention to item No. 88, which is in the middle of page 7. Have you located that?

A. Yes.

Q. And this involved -- this was a letter to the *New England Journal of Medicine,* was it not?

A. That's correct.

Q. And it addressed the subject of air pollution and mortality?

A. Yes.

Q. And you were, in fact, critical of -- in much the manner you've testified today, were you not, of the association between air pollution and mortality?

A. That was a specific criticism, as I recall, of the Harvard Six Cities Study, the first published version of the Harvard Six Cities Study.

Q. And was that not your first publication in the field of air pollution and mortality?

A. Yes.

Q. Let me draw your attention next to item No. 90 on the same page.

A. Yes.

Q. And this is an article that you published, did you not, along with Mr. Luebeck, Mr. Hall and Elizabeth Anderson; is that correct?

A. Yes. Dr. Luebeck, Dr. Hall and Dr. Anderson, yes.

Q. My apologies. You're right.

And that's the same Dr. Anderson who is an expert also in this case; is that correct?

A. That's correct.

Q. And I want to -- this was a reanalysis, was it not, of the Steubenville data?

A. Yes.

Q. And is it not, in fact, the case that this -- that there was funding provided for this article by the American Iron and Steel Institute?

A. Yes, that is correct. All the funding sources should be clearly acknowledged.

Q. And it is on the article; is that correct?

A. I would imagine so, yes.

Q. And I believe your next article that reflects air pollution and mortality is item No. 95, publication No. 95, which begins at the top of page 8 of your deposition. Is that correct? Excuse me. Of your CV.

A. 95, yes.

Q. And this one is also authored by you, Drs. Luebeck, Hall and Anderson; is that correct?

A. Correct.

Q. And it's entitled *Air Pollution and Daily Mortality in Philadelphia;* is that right?

A. Yes.

Q. Is it not also the case that this article was funded by the American Iron and Steel Institute?

A. It probably was. Again, that would be clearly acknowledged on the paper.

MR. GULICK: May I approach the witness, Your Honor?

THE COURT: Yes.

BY MR. GULICK:

Q. Is this the same article we're talking about?

A. Yes.

Q. And this is Plaintiff's --

MR. GULICK: Your Honor, this is marked as Plaintiff's Exhibit 535 for identification.

THE COURT: All right.

BY MR. GULICK:

Q. Dr. Moolgavkar, you indicated this is the article we're talking about, item 95 on your CV?

A. Yes.

Q. Draw your attention to the bottom left-hand corner. There is a series of acknowledgments. Do you see that?

A. Yes.

Q. And you see where it says, "This research was supported by the American Iron and Steel Institute"?

A. Yes.

Q. I believe your next publication, Dr. Moolgavkar, was item No. 98, which is appears to be a letter by you, Drs. Luebeck, Hall and Anderson, and it appears to be a letter to the editor of *Epidemiology*; is that right?

A. Yes.

Q. And it's "Particulate Air Pollution and Mortality." Is that right?

A. Yes.

Q. And then the letter is not sponsored or funded by anyone.

A. Probably not.

Q. And your next publication, Dr. Moolgavkar, on the subject of air pollution is item No. 99 on page 8. Is that correct?

A. Yes.

Q. And this is a critical -- is it not entitled by -- this article is by you and Dr. Luebeck, I believe.

A. Yes.

Q. And it's entitled, *A Critical Review of the Evidence on Particulate Air Pollution and Mortality*, and it's published in *Epidemiology*?

A. Yes.

Q. And it was published in 1996; is that correct?

A. That's correct.

Q. And is it not the case that this article -- that this particular article was also supported by the American Iron and Steel Institute?

A. It probably was, if it says that on the paper.

MR. GULICK: If I could approach the witness, Your Honor?

THE COURT: All right, sir.

BY MR. GULICK:

Q. Now, Dr. Moolgavkar, is this Plaintiff's Exhibit 43, which has been marked for identification as Plaintiff's Exhibit -- excuse me -- Plaintiff's Exhibit 543?

A. Yes.

Q. Is this the article that we have just been talking about by you and Dr. Luebeck?

A. Yes.

Q. I'd like to draw your attention once again to the bottom left-hand corner, Dr. Moolgavkar. Do you see there's some acknowledgments on the bottom left-hand corner of the first page?

A. Yes.

Q. And does it not indicate, among those, "This research was supported by the American Iron and Steel Institute"?

A. Yes.

Q. Dr. Moolgavkar, I believe the next item on this list, that is on your list on your CV that relates to air pollution is item 102, also on page 8. Am I correct about that?

A. Yes.

Q. And this, again, appears to be an article authored by you, Dr. Luebeck, and Dr. Anderson. Is that right?

A. Yes.

Q. And it's entitled *Air Pollution and Hospital Admissions for Respiratory Causes in Minneapolis St. Paul in Birmingham*,

is that right?

A. That's correct.

Q. And it was published in *Epidemiology* in 1997?

A. Yes.

Q. And, Dr. Moolgavkar, is it not also the case that this particular article was supported by the American Iron and Steel Institute?

A. Well, sir, you can see the acknowledgement there. I'm not sure. It's 12 years later, so ...

MR. GULICK: Your Honor, may I approach the witness?

THE COURT: You certainly may.

BY MR. GULICK:

Q. Dr. Moolgavkar, I've just handed you what's been marked as Plaintiff's Exhibit 538, and I will ask you, is this the article which is listed on your CV as item 102 among your publications?

A. Yes.

Q. It is?

A. Yes.

Q. And once again, I'd like to draw your attention to the bottom left-hand corner, Dr. Moolgavkar, and ask if it does not say among the acknowledgements, quote, "This research was supported by the American Iron and Steel Institute," close quote.

A. Yes.
Q. And, Dr. Moolgavkar, I believe the next article on your CV lists that relates to air pollution specifically is item 121. Is that correct?
A. That's correct.
Q. And this was an article that was authored by you and -- is it Dr. Hazelton?
A. Yes. Dr. Hazelton, yes.
Q. And Dr. Luebeck?
A. Yes.
Q. And D. Levy. Is that David Levy?
A. I forget his first name. I think he was a graduate student at the University of Washington. I'm not sure that he received his doctorate by then or not.
Q. And a Dr. Shepherd?
A. Yes.
Q. And this article is entitled *Air Pollution Pollens and Admissions for Chronic Respiratory Disease in King County*; is that right?
A. Yes.
Q. Now, this particular article was funded or supported, in part, by EPA; is that correct?
A. Well, supported, as I recollect, entirely by EPA.
Q. Okay. And then the next item that relates to air pollution is item 122. Is that correct, Dr. Moolgavkar?

A. Yes.
Q. And this is an article authored by you and Dr. Dewanji?
A. That's correct.
Q. And it's called *A Poisson* -- am I pronouncing that correctly?
A. Yes.
Q. P-o-i-s-s-o-n. *A Poisson Process Approach of Recurrent Event Data With Environmental Covariates*?
A. Correct.
Q. And it was published in *EnvironMetric*; is that correct?
A. Yes.
Q. This article also received funding from EPA; is that right?
A. That's correct.
Q. Now, the next article you have regarding air pollution, Dr. Moolgavkar, is item 123, is that right, also on page 9?
A. Yes.
Q. And this article appears to be by you alone; is that right?
A. Yes.
Q. And it's entitled *Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas*.
A. Yes.
Q. And this particular article was also supported by the

American Iron and Steel Institute; is that correct?

A. If that's what the acknowledgement says.

Q. And so the answer is yes?

A. Yes, if that's what the acknowledgement says.

MR. GULICK: May I approach the witness, Your Honor?

THE COURT: Yes.

BY MR. GULICK:

Q. Excuse me.

If you would, would you turn to the last page of this document, Dr. Moolgavkar?

And do you see where it says "Acknowledgements" on the last page of the article?

A. Yes.

Q. And does it not, in fact, say, quote, This work was supported by the American Iron and Steel Institute?

A. Yes.

Q. And I believe your next publication relating to air pollution is item No. 24. Is that correct?

A. Yes.

Q. Now, that's entitled *Air Pollution and Data of Mortality in Three U.S. Counties*; is that right?

A. Yes.

Q. And it was published in *Environmental Health Perspectives* --

A. Yes.

Q. -- in 2000? And is it not the case that this work was supported by the American Iron and Steel Institute?

A. Yes, if that's what the acknowledgement says.

MR. GULICK: May I approach the witness, Your Honor?

THE COURT: Yes.

BY MR. GULICK:

Q. And, Dr. Moolgavkar, is this the article that was entitled -- that was marked which is Plaintiff's Exhibit 539?

A. Yes.

Q. Is this the item 124 on your publications in your CV?

A. Yes.

Q. And I would draw your attention to the bottom right-hand corner of Plaintiff's Exhibit 539. And do you see where it says, quote, This work is supported by the American Iron and Steel Institute?

A. Yes.

Q. And, Dr. Moolgavkar, the next article that relates to air pollution is item number 125?

A. Yes.

Q. Is it by you alone?

A. Yes.

Q. Entitled *Air Pollution in Hospital Admission for Chronic Obstructive Pulmonary Disease in Three Metropolitan Counties*?

A. Three Metropolitan Areas.

Q. I'm corrected. This is published in relation to toxicology?

A. Yes.

Q. I don't have a copy so I can't ask you about it.

After that, is not the next article that you authored for or co-authored on the matter of air pollution item 134?

A. Yes.

Q. And this is another article that you authored with Dr. Dewanji; is that correct?

A. That's correct.

Q. This is *Choice Stratification* -- entitled *Choice of Stratification in Poisson Process Analysis of Recurrent Event Data with Environmental* Covariates? Is that right?

A. Correct.

Q. And this was published in 2002, and you received EPA funding for this; is that right?

A. Well, it was either funded by EPA or not funded at all. It dealt with some ideas that came up during the previous paper that we talked about that was funded by EPA. At this point, I'm not sure whether the funding had run out and we just did it on our own time or whether it was funded by the EPA. The acknowledgements would say that.

Q. Now, Dr. Moolgavkar, before we leave the subject of the American Iron and Steel Institute, is it not in fact the case

that the American Iron and Steel Institute is an industry group?

A. It is. I might point out that all these papers supported by that trade group have appeared in good quality peer-reviewed journals.

Q. I understand. But it is a trade group, is it not?

A. Yes. Yes, it is.

Q. And is it not an entity group that has a financial interest in whether or not fine particulates are associated with human mortality?

A. I don't know that it has a financial interest in whether or not fine particles are associated with mortality.

Q. Is it an industry that survives, Dr. Moolgavkar, on the use of coal as a fuel?

A. It probably does. I don't know what they use as fuel.

Q. Is the next article that you have that relates to air pollution item No. 139, Dr. Moolgavkar, also on page ten of your CV?

A. Yes.

Q. And is it entitled *Air Pollution and Daily Mortality in Two U.S. Counties*?

A. Yes.

Q. And it was published in *Inhalation and Toxicology* in 2003.

A. Yes.

Q. And is it not, in fact, the case that the funding for this particular article was provided by the American Petroleum Institute?

A. It was, if that's what the acknowledgement says. It's always in the acknowledgements.

MR. GULICK: May I approach the witness, Your Honor?

THE COURT: Yes.

BY MR. GULICK:

Q. Dr. Moolgavkar, is this plaintiff's exhibit -- this has been marked as Plaintiff's Exhibit 540 for identification. And I would ask you first, is this the article that we were just discussing, which is item 139, among your publications in your CV?

A. Yes.

Q. And drawing your attention to the bottom of this page, there are some acknowledgements, are there not?

A. Yes.

Q. And among them, does it not say, "Research supported by a contract with American Petroleum Institute-Sciences International, Inc."?

A. Yes.

Q. The American Petroleum Institute is a trade association, is it not?

A. Yes.

Q. Of petroleum companies?
A. Yes. I guess so.
Q. And is it not in fact the case that that industry would have an interest in whether or not air pollution has any association with daily mortality?
A. I guess they have an interest in regulation of air pollution.
Q. And Sciences International, Inc., is that a company with which expert witness Elizabeth Anderson was connected?
A. Yes. I believe she was -- she was president of Sciences International.
Q. And the next article on your CV that relates to air pollution was item 140. Is that right, Dr. Moolgavkar?
A. Yes.
Q. I apologize. And this one is entitled *Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties*. Is that right?
A. Yes.
Q. And this was an article that was requested by the Health Effects Institute, was it not?
A. That's correct. About the time that problems were found with the most commonly used statistical software for analysis of these studies, the Health Effects Institute picked out some studies that it considered particularly important on the issue, which in their review included some of the studies

funded by these trade associations, and asked me to do a reanalysis correcting for the statistical problems, and that is what I did. And I don't believe there was any funding for that study.

Q. And I'd asked you if it was requested by them.

A. I beg your pardon?

Q. I had asked you if it was requested by the Health Effects Institute.

A. Yes. It was requested by the Health Effects Institute, yes. But I might point out that it was a request to reanalyze what they considered important studies, which included the studies that you have just gone through supported by trade groups.

Q. And, Dr. Moolgavkar, the next article among your articles in your CV that relates to air pollution is item 149. Is that right?

A. Yes.

Q. And it was authored by you alone, it appears.

A. Beg your pardon?

Q. You were the only author of this article, weren't you?

A. That's correct, yes.

Q. And is it not entitled *A Review and Critique of the EPA'S Rationale for a Fine Particle Standards*? Is that right?

A. That's right.

Q. And it was published in -- is that *Regulatory Toxicology* --

A. Yes.

Q. -- *and Pharmacology?*

A. Yes.

Q. And that was in 2005?

A. Yes.

Q. And I ask you, Dr. Moolgavkar, whether or not this article received support from the American Petroleum Institute.

A. It did.

Q. I believe the next item on your CV that relates to air pollution is No. 156 on your CV? Is that right?

A. That's correct.

Q. And it was an invited editorial on the Enstrom paper that you referred to earlier in your testimony?

A. Yes, it was.

Q. And being an invited article, there was no funding source?

A. There was no funding for that, no.

Q. And the next item on your list -- the next article on your list that relates to air pollution appears to be some correspondence by you, which is item 159; is that correct?

A. That's correct.

Q. And it appears to be, I guess, correspondence by you to

the *Journal of Nature*; is that right?
A. Yes.
Q. And it's entitled *Pollution Analysis Flawed by Statistical Model*; is that right?
A. Yes.
Q. And being a letter, it was not funded?
A. No. No funding, no.
Q. And the next article on this list is an article by you and Dr. Anderson and Dr. Rice and Dr. Cross, Dr. Hidy, Dr. Hoel, H-o-e-l, Dr. McClellan and you; is that right?
A. Yes.
Q. This was published -- on your CV, it says that it's in press. But this article was since published in 2007; is that right?
A. It has been published, yes.
Q. And the title of this article is *Evidence of Health Impacts of Sulfate and Nitrate Containing Particles in Ambient Air*. Right?
A. Yes.
Q. And it was published in *Inhalation and Toxicology*.
A. I believe so, yes.
Q. And I ask you, Dr. Moolgavkar, whether or not this article was funded in part -- or funded by the Edison Electric Institute and Industry?
A. I believe it's called the Edison Institute. Yes, it

was.

Q. But it is a -- the Edison Institute is, in fact, a trade group of the electric power industry; is that right?

A. That's my understanding.

Q. Have you published other articles since that time, Dr. Moolgavkar, that relate to air pollution?

A. No, I have not.

Q. Thank you.

Also in your CV, Dr. Moolgavkar, you listed some litigation in which you've been a testifying witness; is that right?

A. That's correct.

Q. And those are listed, are they not, at the -- on page 16 of your CV?

A. Yes.

Q. And the first you have listed, in 2006, you have trial testimony, and that was *Carl and Joyce Holbrook vs. Bondex, International?*

A. Yes.

Q. And you were representing an industry party in this case; is that right?

A. I believe it was one or more of the U.S. automobile companies.

Q. And *Rebekeh Price vs. Borg-Warner.* This is in the Superior Court of the State of California. You were also

representing an automobile company; is that right?

A. Yes. One or more automobile companies.

Q. And these were defendants in actions alleging exposure to a cancer-causing agent -- allegedly a cancer-causing agent. Is that right?

A. Yes. These cases have to do with alleged exposure to asbestos from friction products.

Q. And in 2006, you also gave a deposition, did you not, in *United States, et al, vs. American Electric Power and Service Corporation*?

A. Yes, I did.

Q. And were you an expert witness for American Electric Power?

A. It did not go to trial.

Q. Did you give a deposition on behalf of American Electric Power in that?

A. Yes, I did.

Q. And then it appears you also gave a deposition in the *Price vs. Borg-Warner* matter. That's the same case that you testified at trial at?

A. Yes.

Q. And next deposition is the *Holbrook vs. Bondex*. And, again, you were an expert witness for one or more automobile companies; is that right?

A. Yes.

Q. Then, also, in 2006, you testified in the matter of, *Tizcareno*, spelled T-i-z-c-a-r-e-n-o, *vs. Burns International*.

A. Yes.

Q. And is this another series of cases involving automobile manufacturers?

A. Yes.

Q. And you were testifying on behalf of one or more of the automobile manufacturers?

A. That's correct.

Q. And the next listed item is a deposition in *Caroline Hicks vs. American Asbestos Company*; is that right?

A. Yes.

Q. And you were testifying in this case for American Asbestos Company? Or was it another company?

A. No. It was, again, one or more of the automobile companies.

Q. So this was also in that series of litigation; is that right?

A. Yes.

Q. And finally, you gave a deposition in 2006 in the matter of *Halsema*, H-a-l-s-e-m-a, *vs. Allied Packing*?

A. Yes.

Q. And was this another in that series of litigation?

A. For one or more of the automobile companies, yes.

Q. And that was your client?

A. Yes.

Q. And then, in 2005, you also gave a deposition in the matter of *Samuel Gates vs. A-1 Clutch Company*. Is that right?

A. Yes.

Q. And once again, you were an expert witness for one or more automotive companies in that matter; is that right?

A. Yes.

Q. And then you also gave a deposition in the matter of *Louis Zavacky*, Z-a-v-a-c-k-y, *vs. Dana Corporation, et al*, in Ohio?

A. Yes.

Q. And you were an expert witness for the defendant in this matter, were you not?

A. Yes. Again, one or more of the automobile companies.

Q. So this was a similar action about the same general subject matter but in another state?

A. Yes.

Q. Then, in 2004, you gave a deposition in the matter of *United States, et al, vs. Ohio Edison, et al.* Is that right?

A. Correct.

Q. And in that deposition, you were serving as an expert witness for Ohio Edison; is that correct?

A. That's correct.

Q. In 2002, you gave a deposition in the case of *United States, et al vs. W.R. Grace;* is that right?
A. That's right.
Q. And you were an expert witness for W.R. Grace; is that right?
A. Yes.
Q. And there were two of these matters that involved coal-fired power plants; is that correct?
A. That's right.
Q. And those were *United States, et al vs. American Electric Power* and the *United States, et al vs. Ohio Edison;* is that correct?
A. Yes.
Q. Now, I believe, Dr. Moolgavkar, that during your deposition, did you not state that you were agnostic -- I think that was your word -- about whether PM2.5 exposure could cause adverse health effects? Is that correct?
A. That is correct. That is exactly what I said today.
Q. And did you not state that even though you agree there is a substantial volume of literature showing associations between air pollution and adverse health effects, adverse effects on human health? Is that also correct?
A. There is a substantial body of literature on both sides of the issue.
Q. But you did acknowledge, did you not, that there is a

substantial volume of literature showing associations between air pollution and adverse health effects?

A. Well, there is a substantial volume of literature on both sides. There's clearly a substantial volume of literature showing associations.

Q. So the answer is yes?

A. Yes, the answer is yes.

Q. And is it not, in fact, the case that there have been both long-term and short-term studies that, in fact, found correlation between PM2.5 -- excuse me -- long-term PM2.5 exposure to premature mortality?

A. Yes, there are.

Q. Is it not, in fact, the case that recent research has bolstered the evidence for the biologically plausible pathways in which PM2.5 could cause adverse cardiovascular effects. Is that correct?

A. As I said, I'm not a toxicologist, particularly in that area, but I am not convinced that there is any evidence bolstering the biological plausibility.

Q. But you're not an expert in that area?

A. I'm not an expert. I just follow the literature from the outside.

Q. Is it not, in fact, the case that you have no opinion on whether, if PM2.5 exposure could, in fact, cause premature death, that there is a threshold ambient concentration below

which there is no effect?

A. I believe there is not enough epidemiological information to either support or refute the existence of a threshold. We simply don't have the data to determine that, if one makes the assumption that the effect is causal in the first place.

Q. At your deposition, did you not agree that the control of fine particulate matter may have benefits for public health?

A. I did say "may have benefits for public health," yes.

Q. So the answer is yes, right?

A. The answer to that question as raised is yes.

Q. And you also agree that it is entirely reasonable to regulate air pollution on the basis that control of pollutants may have public health benefits, correct?

A. Yes, I believe I said on the basis of a precautionary principle, yes.

Q. Now, you recall that at your deposition, Dr. Moolgavkar, we took a look at the -- we took a look at the -- your expanded expert judgment assessments of the concentration response relationship between PM2.5 exposure and mortality?

A. Yes, we did.

Q. Excuse me. I have too many things in front of me.

Dr. Moolgavkar, I'd like to draw your attention to Plaintiff's Exhibit 242.

MR. GULICK: And with Your Honor's permission, I'm going to go ahead and find this volume up there for him.

THE COURT: All right, sir.

MR. GULICK: And, Your Honor, it is plaintiff's notebook No. 4.

THE COURT: I have that.

BY MR. GULICK:

Q. Now, Dr. Moolgavkar, you are familiar with this document, are you not?

A. Yes, I've seen it.

Q. And, in fact, this is entitled *Expanded Expert* -- this is Plaintiff's Exhibit 242. It's entitled *Expanded Expert Judgment* -- excuse me -- *Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between PM2.5 Exposure and Mortality*. Is that right?

A. Yes.

Q. And this is the final report?

A. Yes.

Q. Published September 21, 2006?

A. Right.

Q. Is that right?

I'd like to draw your attention, if you would, to page 6. If you'd look at the top of the page, Dr. Moolgavkar, you will see that there is a printed -- printed in blue at the top of the page, you'll see it's page 6 of 109.

A. Yes.
Q. Have you found it?
A. I see it.
Q. And I wanted to ask you Dr. Moolgavkar, is Daniel Krewski the author of the report that you were referring to earlier?
A. Yes.
Q. He is the same Daniel Krewski with whom you have published articles; is that right?
A. Yes. He's a member of the panel.
Q. Joe Mauderly? Am I pronouncing his name correctly?
A. Joe Mauderly, yes.
Q. M-a-u-d-e-r-l-y?
A. Yes.
Q. Is this the same Joe Mauderly that invited you to participate in CASAC?
A. Yes.
Q. But you declined.
A. I declined, yes.
Q. And you are also familiar with most all of these other persons that are named on this final expert list; is that right?
A. Yes. I believe I know all of them personally, with the exception of Nino Künzli?
Q. And for the sake of the court reporter, that's spelled

K-u, with an umlaut over it --
A. Yes.
Q. -- n-z-l-i?
A. Correct.
Q. I'd like to draw your attention, Dr. Moolgavkar, to page 11 of this same document. It says page 11 of 109 at the top.
A. Yes.
Q. Excuse me. I went one page too many. Page 10.
And here, in bullet form, does it not list a set of conclusions, Dr. Moolgavkar?
A. Yes.
Q. And the first sentence of the first conclusion says: "The experts in this study tended to be confident that PM2.5 exposure can cause premature death"?
You don't agree with that, do you?
A. Well, I agree with the statement that experts tended to be confident. I agree with that statement.
Q. No, I mean --
A. I don't agree --
Q. -- you don't agree with their conclusion?
A. -- with the conclusion.
I might point out that in the pilot phase of this study, three of the five experts were skeptical of the association.
Q. In fact, the pilot study is what you listed in your second supplemental report, did you not?

A. That's the one I referenced in my second supplemental report.

Q. You did not reference the final report, did you?

A. I think I referenced the final report.

Q. But you didn't discuss its results.

A. I discussed some of its results. I'd have to go back to my report to see exactly what I said.

Q. Well, you actually discussed this particular study in your supplemental report; is that right?

A. Which specific study? I have discussed each one of these studies, Jarrett, Laden, and Sonval (phonetic) in one or another of my reports.

Q. No. I meant the expert -- this expanded judgment document we're looking at now.

A. I'm sorry. I don't understand the question.

Q. The document you're looking at right now.

A. Yes.

Q. This exhibit.

A. Yes.

Q. This particular -- this whole document we're looking at right now, which is Defendant's Exhibit 242, that's -- this you did not discuss in your first report, did you?

A. Not in my first report, no.

Q. But you discussed it in your supplemental report.

A. I believe I did.

Q. And does it not state in the second sentence of this, that same bullet point, that 10 of 12 experts believe that the likelihood of a causal relationship was 90 percent or better? Or higher. Excuse me.

A. Yes.

Q. I know that's what it says. But you don't agree with their belief as expressed in this sentence, do you?

A. Well, I'm in agreement with at least two of the 12 experts who did not agree with this conclusion.

Q. However --

A. And with the three experts.

Q. That's in the next sentence, is it not, Dr. Moolgavkar, that, quote: The remaining two experts gave causal probabilities of 35 and 70 percent.

A. I would tend to be at the 35 percent level.

Q. You are at the 35 percent level?

A. Possibly, yes. It's very difficult to quantify the personal probabilities of the studies.

Q. Was this expert elicitation not conducted entirely for the purpose of characterizing the uncertainties associated with the question in connection between PM2.5 and premature mortality?

A. That's what the elicitation was conducted for. However, I don't think it was properly done.

Q. The second bullet on the same page, Dr. Moolgavkar, does

it not say that only one of 12 experts explicitly incorporated a threshold into its CR -- and that stands for concentration response, does it not?

A. That's what this states.

Q. CR function.

A. That's what it states, yeah.

Q. And the rest -- doesn't the next sentence say, "The rest believe that there was a lack of emperical and theoretical support for population threshold"?

A. That's what it says.

Q. You do not agree with that either?

A. I think I said today there is no evidence to assert the existence or non-existence of a threshold, and, to me, that sentence says the same thing. There is no evidence to support a threshold or not to support a threshold.

Q. In the next bullet, which is the last bullet on this page, does it not say that "The experts relied upon the course of cohort epidemiologic" -- let me start again.

"The experts relied on a course of cohort epidemiology studies to derive their quantitative estimates, mainly those associated with the ACS and the Six Cities cohorts." Is that correct?

A. That's what it says, yes.

Q. And does not the third sentence of this say, quote: The greater emphasis on Six Cities appeared to result from

corroborating evidence in the recent Six Cities follow-up by Laden, et al, in 2006?

Is that right? Is that what it says?

A. That's what it says, yes.

Q. Is that the same Laden that you were referring to in your earlier testimony?

A. Yes.

Q. In fact, Dr. Moolgavkar, is it not the case that you agree with the clear majority of the scientists who were involved in this expert elicitation? Is that right?

A. I beg your pardon?

Q. You disagree, do you not, with the clear majority of scientists who were involved in this expert elicitation?

A. Disagree about what, Mr. Gulick?

Q. The causal relationship between PM2.5 exposure and premature mortality.

A. Yes. I'm of the opinion that there isn't sufficient evidence to infer a causal relationship at this time.

Q. During your testimony, Dr. Moolgavkar, you mentioned an article by -- or actually you were asked about an article by Pope and Dockery that was published in *Air and Waste Management Association* in 2006 entitled *Health Effects of Fine Particulate Air Pollution Lines That Connect*.

A. I believe counsel read out portions of that paper, yes.

MR. GULICK: Your Honor, this article is actually

attached as an attachment to Dr. Peden's expert report, as counsel indicated, but rather than hunting around, I'm going to hand a copy to the witness, with your permission, as well as to the Court.

THE COURT: All right, sir.

BY MR. GULICK:

Q. Dr. Moolgavkar, I've just handed you what was marked in your deposition as Plaintiff's Exhibit 8. Was that the article that we've just been talking about?

A. Yes.

Q. Let me draw your attention to the abstract. About not quite two-thirds of the way down, there are the words -- there is a sentence and it says: "Despite important gaps in scientific knowledge and continued reasons for some skepticism, a comprehensive evaluation of the research findings provides persuasive evidence that exposure to fine particulate air pollution has adverse effects on cardiopulmonary health."

A. I see it.

Q. Do you see it?

A. Yes.

Q. You do not, in fact, agree with that conclusion, do you?

A. Well, first, I'm assuming they mean fine particulate air pollution at current ambient levels in western Europe and American cities, because, without that qualification,

clearly, if you have very high levels of particulate pollution, they would have adverse effects on cardiopulmonary health.

Q. I'm sorry. I didn't hear what you just said.

A. At very high levels, very high concentrations, I would imagine there would be adverse effects on cardiopulmonary health. But I do not agree with this statement here that ambient levels of air pollution or particulate, fine particulate air pollution in the United States, that there is convincing evidence that it has adverse effects on cardiopulmonary.

Q. Doctor, I'd like to take you to the end of this article, on page 721. Article number is listed at the bottom of the page on the right.

Have you found that page?

A. Page 721?

Q. I'm sorry. 29. 729.

A. Yes, sir. I have it.

Q. I'm going to draw your attention to the paragraph that begins the text on this page, and almost in the middle of that paragraph there is a sentence that says, "There are almost certainly multiple mechanistic pathways with complex interactions and interpendencies." Do you see that sentence?

A. Yes, sir, I see it.

Q. And this is talking about the mechanistic pathways by

which exposure to fine particulates could cause cardiovascular effects; isn't that right?

A. With all respect, Mr. Gulick, that sentence basically says nothing. It just says there are multiple mechanistic pathways with complex interactions and interdependencies. You can say that about any biological phenomenon.

Q. Then I'm going to take you to the last sentence. It begins with the words: "Although much remains to be learned."

You see where it says that?

A. Yes, I see that.

Q. That sentence says, quote: "Although much remains to be learned, it is no longer true that there are no known pathophysiological or mechanistic pathways that could plausibly link PM exposure to cardiopulmonary disease and death."

A. Well, that again sounds like a very general statement to me. It doesn't say anything about specific pathways, and, besides which, you know, as I said, I'm not a toxicologist and I can't comment on individual pathways of disease.

MR. GULICK: Your Honor, I notice what the time is. I think I only have a few more questions, and I can probably bring it shorter if I can look at this a little bit during a break, if that's all right with you.

THE COURT: You think it might shorten it to take a

break?

MR. GULICK: Yes, sir.

THE COURT: We'll indulge that and take a break.

MR. LANCASTER: No objection from defense.

THE COURT: Take a recess.

(Recess.)

THE COURT: All right. Mr. Gulick?

MR. GULICK: Your Honor, I was unable to get it down to no more questions, but I'm down to the last little bit.

THE COURT: Go ahead.

MR. GULICK: May I approach the witness?

THE COURT: Yes.

MR. GULICK: Thank you, Your Honor.

BY MR. GULICK:

Q. Dr. Moolgavkar, I have just handed you a document that has been marked as Plaintiff's Exhibit 492. Do you have it?

A. I have it here, yes.

Q. And this was Exhibit No. 5 to your deposition, so you have seen this before at the deposition.

Do you remember that?

A. Well, I recall being shown it, yes.

Q. And I'm going to ask you if this is not -- does it not say at the top that this is part of the *Federal Register* at the top?

A. Yes.

Q. Volume 70, No. 091? Is that right?

A. Yes.

Q. And a little bit below that it says, quote: "Rule to Reduce Interstate Transport of Fine Particulate Matter and Ozone (Clean Air Interstate Rule)." Do you see that?

A. Yes.

Q. Now, as I did before, Dr. Moolgavkar, this is a portion of a very long document, so I'd like you to turn just a few pages to what has at the top right-hand corner, it says page 285, even though it's only one page over. Do you see that?

A. Page 285?

Q. Yes. It's only one page in, in fact, on this because this is a part of a long document.

A. Yes.

Q. It also says at the top, right in the center, "70FR25162," and then an asterisk, correct?

A. Yes.

Q. Do you see in the middle of this page that it indicates *Human Health Benefit Analysis* as a title?

A. I see that.

Q. And does not the very first sentence say: "Our analysis of the health and welfare benefits anticipated from this rule are presented in this section"?

A. That's what it says.

Q. And then it says briefly, "The analysis projects major benefits from implementation of the rule of 2010 and 2015." Is that right?

A. That's what it says.

Q. It then refers to X-1 in the next paragraph. See where I'm talking about, the paragraph?

A. Yes. Table X-1. Yes.

Q. Okay. I'm going to read to you the second sentence of this paragraph:

"In 2015, we estimate the PM-related annual benefits include approximately 17,000 fewer premature fatalities, 8,700 fewer cases of bronchitis, 22,000 fewer non-fatal heart attacks, 10,500 fewer hospitalizations for respiratory and cardiovascular disease combined, and resulting in significant reductions in days of restricted activity due to respiratory illness (with an estimate of 9.9 million fewer cases) and approximately 1,700,000 fewer work-loss days."

Did I read that sentence correctly?

A. Yes, you did.

Q. And, indeed, Table X-1 can be found if you turn the page once more and look at what's marked at the top of page 287. Have you found that page?

A. It's coming up on my screen, yes.

Q. Okay. And what I want to draw your attention to with respect to these findings -- this was the table that was

referred to, was it not, in that previous paragraph?
A. I believe it is.
Q. What I want to draw your attention -- Doctor, you had mentioned early in your testimony that you felt that there was something wrong with the estimates of Dr. Levy and that his estimates of premature mortality exceeded in number the hospital admissions for respiratory and cardiovascular disease. Did you not?
A. Yes, sir, I did.
Q. Is that not in fact the result, Dr. Moolgavkar, of the fact that short term -- that Dr. Levy used short-term studies for his concentration-response function of hospital admissions, whereas if he had used long-term studies --
A. Mr. Gulick, no matter whether Dr. Levy does it or the USEPA does it, no matter whether short-term study they used or long-term study they used, these numbers are being presented as actual numbers of the benefit that would accrue -- of the benefits that would accrue from lowering air pollution, and if the statistical methods produce numbers that fly in the face of common sense, then the statistical numbers have to be questioned. And it does not matter whether Dr. Levy does it, doesn't matter whether EPA does it, and doesn't matter whether you use short-term studies or long-term studies. These numbers, if they are to be taken seriously, they have to meet a very simple test. They don't

pass that test.

Q. Well, Dr. Moolgavkar, is it, in fact, not the case that hospital admissions from time-series studies only capture today's pollution effects on tomorrow's hospital admissions?

A. That may well be true.

Q. So this is, in fact -- they are, in fact, underestimating the total hospital admissions?

A. These are being presented, as are the numbers presented by Dr. Levy, as a correct representation of what would happen. Nowhere is it stated that this is not a correct representation.

Q. Dr. Moolgavkar, in fact, these are their best estimates of those figures?

A. These -- all these estimates presented here are probably best estimates. I haven't done the calculations, so I don't know whether the calculations are correct or not. But these are best estimates under a set of assumptions. You have to accept those assumptions before you can agree with the estimates.

Q. If the estimate is that there is definite hospitalization, Dr. Moolgavkar, based on a short-term series, or short-term study, would it not, in fact, be the case that if air pollution causes one to develop cardiovascular disease and you are later hospitalized for it, that that would not be captured in the short-term study?

A. Well, these numbers are being presented as serious estimates of the number of events that would be prevented; and no matter where they come from, these numbers cannot be taken seriously in view of this strange feature that the hospital admissions is smaller than the number of deaths.

Q. In fact, is it not the case that the hospital admissions are, in fact, conservative estimates because they use the time-series study?

A. It might be the case that the mortality figures are exaggerated.

Q. Would you answer the question, please?

A. Well, I mean, basically you're asking me, Mr. Gulick --

Q. I'm asking you about the time series.

A. -- why the hospital admissions numbers are smaller than the mortality numbers, and all I'm telling you is that it could be because both the set of numbers are meaningless because the assumptions underlying them are meaningless. It could be because the hospital admissions are underestimated or it could be because the mortality figures are overestimated. All of those three scenarios could give rise to numbers of this type.

Q. And in fact, Dr. Moolgavkar, you disagree both with Dr. Levy and the USEPA on this subject.

A. Well, the USEPA is an agency, and they have put these numbers together on the basis of assumptions about dose

response relationships. These numbers do not indicate causality. These are numbers that the USEPA is required to produce to talk about the cost benefit analysis of any kind of regulation it is proposing, and it is based on a set of assumptions that I have questioned, and I have given you reasons as to why I questioned those assumptions.

Q. In fact, they are findings and estimates that they've made, is that not the case, Dr. Moolgavkar, not assumptions?

A. I'm sorry?

Q. In fact, they're the findings of the EPA; is that right?

A. These are findings of EPA based on a set of assumptions?

Q. And you disagree with them?

A. Yes. And I spent the better part of an hour explaining why I disagree with those assumptions.

MR. GULICK: Thank you.

I have no further questions. I do want to move into evidence Plaintiff's Exhibit 492.

THE COURT: All right. I'll let that be admitted.

MR. GULICK: I have no further questions.

(Defendant's Exhibit 492 received in evidence.)

MR. LANCASTER: I have no redirect, Your Honor.

THE COURT: No redirect?

All right, Dr. Moolgavkar, that will complete your testimony and you are excused.

Mr. Lancaster?

MR. LANCASTER: Defendant TVA calls as its next witness Dr. Elizabeth Anderson.

ELIZABETH ANDERSON,

being duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

MR. LANCASTER: Your Honor, we're still using defendant's book 14. I would ask Dr. Anderson to retrieve book 14 from the shelf.

THE COURT: All right.

BY MR. LANCASTER:

Q. Could you please state your full name for the record.

A. Yes. My name is Elizabeth L. Anderson.

Q. And where do you live, Dr. Anderson?

A. I live in Alexandria, Virginia.

Q. And you've been retained by Tennessee Valley Authority as an expert witness in this matter?

A. Yes, I have.

Q. And you've offered two reports that I've marked for identification as TVA Exhibit 345 and 346.

A. Yes, I have. One dated February of '07 and the other June of '07.

Q. And would you please tell the Court about your education.

A. Yes. I attended undergraduate college at the College of

William and Mary, where I was a premedical student taking equal credits in biology and chemistry.

I ultimately decided to go accept a fellowship to the University of Virginia rather than medical school, where I concentrated on mechanistic organic chemistry and taught the undergraduate program for the premedical school laboratory --

Q. If you wouldn't mind speaking up. I'm having a little trouble hearing.

A. Okay. I'm sorry. I apologize. I have a problem with my throat.

Premedical school students, a laboratory in organic chemistry.

Q. And did you finish describing your education?

A. No. I didn't. Then I received a fellowship from the Defense Department to attend -- to get my Ph.D., but the program dictated that I be in a program where I could do my research at a military institution. I subsequently continued my work in mechanistic organic chemistry, achieving my Ph.D. from American University.

Q. And your Ph.D. is in organic chemistry?

A. Mechanistic organic chemistry.

Q. And you are, in fact, a toxicologist.

A. Yes. At the time I got my degree, there was no degree in toxicology.

Q. And once you obtained your doctoral degree, that was

about the same time EPA came into existence, wasn't it?
A. Yes, it was. It was a very fortunate time for me, because I had finished a year's of research that was required under my fellowship, so that I could then pursue my interest in human health applications. And my background had been in mechanistic organic chemistry, that is, the science of designing molecules to make them effective or ineffective for human purposes, for example, for pharmaceutical applications, so the opportunity to join EPA and look at the alternative side of that issue of how can toxic chemicals interact with humans when they're exposed to cause harm was a very appealing opportunity.
Q. All right. And what were your first duties at the EPA?
A. My first assignment was to be a member of a small technical team. I worked directly with the administrator, Bill Ruckelshaus, the first administrator of EPA, and the general counsel, John Quarles, in the earliest technical cases that required scientific analysis, but to address some of the most important pollution issues in the United States in the first few years of EPA's existence.
Q. And then did you become a science adviser?
A. Yes. Subsequently, after the first three years, I became the Deputy Assistant Administrator for the Air Programs as a scientist, assistant administrator of the attorney, and it was my responsibility, particularly, to

focus on the Ambient Air Quality Criteria Standards and the underlying science, and I spent a year in that assignment. And then, right after that period, I was asked then to be the executive director of a committee to address the fact that EPA was having great difficulties with its regulatory policies, which at the time had sought to achieve a zero risk for any agent that was thought to cause cancer in humans or animals.

This goal had been taken from the Delaney Clause, the Food, Drug and Cosmetic Act, where intentional food additives had this requirement that there be zero risk for any agent that was found to cause tumor in animals or humans. And, in fact, that had been transferred to EPA, and the expectation was that EPA would be able to achieve the same zero risk.

We, in fact, found that that was impossible. In the first few years at EPA, the actions that we had taken had brought headline news to the agency, and in a very negative way, because we had been unable to achieve zero risk for agents, such as benzene in gasoline, benzene being a well-characterized carcinogen. At the same time, we had to account for very economically important pesticides simply on the basis of the animal, too, with no regard for the response in tech levels or exposure levels.

So my committee was asked to write the cancer policy for the agency. In the early part of the '70s, there was a

feeling, a sense in the country, that there was an epidemic of cancer and that that epidemic was perhaps largely caused by environmental agents. So there was a great deal of attention on this particular early effort at EPA.

Our committee decided that we could not write a cancer policy but, rather, we reported out a process, and that process was a two-step process. It was the first time that any federal agency had adopted risk assessment to address the health effects of environmental agents. We reported at that policy. The first step was to perform a risk assessment. The second step was to determine how much risk would be acceptable, the risk management step. In the risk assessment step, we formulated two questions. How likely is an agent to cause harm; and on the assumption it can cause harm, how much harm can it cause quantitatively, and it was the first time that any federal agency had really attempted to concentrate on the enormous burden of characterizing dose response relationships, that is, how much exposure to an agent would cause harm, either given epidemiology studies or animal studies, and then to associate that relationship with exposures.

I was a co-author of the scientific paper that was the guidelines published in the *Journal of the National Cancer Institute,* which was a part of this policy that was announced at that time.

Subsequently, I was asked to form the Carcinogen Assessment Group, which was called for by the guidelines that the agency would need to have a scientific body to carry out the responsibility of the risk assessment programs. I formed the Carcinogen Assessment Group. And then I formed the expanded components of the risk assessment programs at EPA for other health effects and the two criteria document offices, one that was responsible for the ambient air quality criteria documents and the other for the water program support documents.

I spent 14 years at EPA. Ten of those years I spent directing the central risk assessment activities of EPA. And for my work at EPA, they gave me their highest award, their Golden, for leadership in these programs.

Q. And while you were leading and directing EPA's Central Risk Assessment for about a decade, did you perform any toxic risk assessments?

A. Yes, I did. I was very actively involved in the risk assessment programs.

I co-authored literally hundreds of risk assessments during this period, for the air programs, for the water programs, for pesticides, and for the for toxic substances programs once they were -- once that act was passed, and for -- and we also assisted the hazardous waste, the superfund programs.

This office was responsible for either doing all of the risk assessments or reviewing risk assessments done anywhere else in the agency.

And then, in the final regulatory packages -- at that time we called them red border review packages -- I had the final sign-off authority for the quality of the science in those regulations.

Q. And Doctor --

May I approach the flip chart, briefly, Your Honor?

THE COURT: All right.

BY MR. LANCASTER:

Q. Let me make sure I have it on the right page.

Dr. Levy drew a flow chart that's been marked as Plaintiff's Exhibit 485 of a methodology that he says dates back to something he called the Red Book.

Do you have any familiarity with the Red Book?

A. Yes, I certainly do. The Red Book is the landmark publication that came from the National Academy of Sciences, the National Research Council in 1983. It was largely stimulated by our work at EPA. Since no other federal agency had attempted doing risk assessment, which we had borrowed from a long history of risk assessment applications in other areas, to nuclear power plants, bridges, dams and engineering areas, and radiation. There was close scrutiny to monitor what EPA was doing and how this whole risk assessment program

was really working.

The National Academy was called upon to assemble this committee that reported as this Red Book, largely because of the efforts that had been under way at EPA from 1976 until the early '80s, when this committee was formed.

I regularly advised the committee. We had performed 100 -- about 150 risk assessments at the time this committee met, and the committee was very interested in how EPA's risk assessment programs had been operating. So I was very just involved with the committee and knew the work of the committee quite well and know the results of the Red Book. The Red Book reported on both the paradigm -- it was a process -- and a number of other very important findings calling for guidelines which mimicked the program at EPA. The four steps mimicked that, two questions, such as combining the quantitative question, the dose response and exposure. But they mimicked our risk assessment management, and, in large part, it endorsed what we had been doing since 1976.

Q. And then, in the mid 1980s, you left EPA?

A. Yes. I left EPA in 1993, I believe. No. I'm sorry. I left EPA at the beginning of 1986, and continued doing very much the same kind of work in the private sector as I had been doing at EPA, from a scientific standpoint of course.

Q. And is risk assessment what you continue to do to this

day?
A. That's right. I continue my work in risk assessment. I am currently vice president to health sciences at Exponent. Our group has five centers: Epidemiology; a center for toxicology; a center for exposure assessment; a center that is industrial hygiene and public health; and a final center that addresses food safety, nutrition, and chemical regulation. We have 15 offices in five countries.
Q. Do you hold any professional organization memberships?
A. Yes, I do. I think the most important involvement I've had for a very long time is with the Society for Risk Analysis. I was a founding member of that society. I served as their president. I served on their council. I've served -- chaired numerous committees. I'm a fellow of the society. And I have, for the last ten years, been the editor-in-chief of the flagship peer-reviewed journal of the society, without a doubt the most important journal internationally that addresses topics in risk assessment. This journal is circulated in over 80 countries and it has, I think, greater than a 4,000-member institutional subscription level and then many individual subscriptions.
Q. And did you receive an award from the society for risk analysis in 2006?
A. Yes, I did. The society has given me a couple of awards, but I think the most important one was their very

unique honor, and they don't give this award very often: The presidential award for the service of being editor-in-chief for the journal.

Q. And are you a member of any toxicological or other scientific associations?

A. Yes, I am. I am a member of the Society for Toxicology; I'm a fellow of the Academy of Toxicological Sciences; a member of the American College of Toxicology; I serve on the board of trustees for the Toxicology Education Foundation, and I'm a member of several other organizations. I'm on the editorial board of other journals.

Q. And have you published in your field?

A. Yes, I have. I've published many articles in the field of risk assessment on different topics in the field.

Q. And have you been an invited speaker at any colleges or universities?

A. Yes, I have. I have lectured at most major universities in the United States and many abroad. I've served on numerous international panels: The World Health Organization, the Pan American Health Organization, and have lectured for them and conducted course work for them.

Q. And have you ever testified as an expert witness before?

A. Yes, I have. At EPA, I was involved in administrative hearings. I testified on Capitol Hill a number of times while at EPA. I've been invited to testify on Capitol Hill

since I've left EPA. And I've testified in a few legal proceedings.

Q. And in this case, has EPA -- excuse me -- has TVA asked you to evaluate the potential health impacts of emissions from TVA power plants in the state of North Carolina?

A. Yes, they have.

Q. And have you done that?

A. Yes. Yes, I have.

Q. And are your conclusions documented in your written reports that you previously identified?

A. Yes, they are.

MR. LANCASTER: Your Honor, defendant tenders Dr. Anderson as an expert in the fields of toxicology, risk assessment, and integrating interdisciplinary sciences for risk assessment, which I understand from a brief conversation with Mr. Gulick, is not -- there is no objection.

MR. GULICK: I do not object, Your Honor, to the -- the State does not object to the stated expertise as was stated.

THE COURT: All right. Let the stipulation be recorded as stated.

MR. LANCASTER: At this time, Your Honor, I would also move into evidence TVA Exhibits 345 and 346, Dr. Anderson's reports.

MR. GULICK: Your Honor, we have the same objection

as before.

THE COURT: All right. Objections are overruled.

(Defendant's Exhibits 345 and 346 received in evidence.)

MR. GULICK: Thank you, Your Honor.

THE COURT: Yes.

BY MR. LANCASTER:

Q. Dr. Anderson, the Court has heard extensive testimony from Mr. Wheeler, Mr. Chinkin, and Dr. Tesche about the air dispersion modeling that was performed in this case to estimate air quality impacts in North Carolina from TVA's plan.

Did you base your analysis on the output of those models?

A. Yes, I have.

Q. And was there a particular health end point on which you focused most of your attention?

A. Yes. I focused particularly on the particulate matter end point, and particularly mortality. And this is because, in Dr. Deck's analysis, taking the output of Dr. Spengler and Levy's report, he has estimated that 98 percent of the benefits are from reductions in particulate emissions or particulate impacts, and only 2 percent for ozone. And of those, he's projected approximately 95, 96 percent for the mortality. And I noted particularly that morbidity, other

health impacts other than mortality were very, very small percentages; for example, asthma, .1 percent. So I have focused particularly on the 95, 96 percent benefit part of that analysis, meaning I have looked particularly at particulate matter and the mortalities.

Now, in my report, I discuss extensively other aspects as well as ozone, but I think the most focus is on mortality and particulate reductions.

Q. Thank you.

And there is an awful lot of data that comes from this computer modeling. Have you made a summary of some of the data that relates to your specific analysis?

A. Yes, I have. And you're correct, there really -- I received a lot of information. The information that I have in my reports result from the modeling, the CMAQ modeling for the year 2002 that could present emissions by, I understand something called zeroing out, for each facilitate that TVA owns of the 11 plants.

I also received CAMX data with two grids, a smaller grid of 12 and a larger grid of 36, for 2002, and then the same information for particulate matter for 2013. And in 2013, I received two outputs. I received the baseline -- what I'm calling baseline -- of what TVA projects its emissions to North Carolina would be in 2013.

I also received output that would allow me to look at

the difference between that level and how much additional benefit, or how much additional reduction there would be, if TVA were to adopt the additional controls that I understand are associated with North Carolina's Clean Smokestacks Act.

Q. And that's the concept that I believe Dr. Levy described as "Delta."

A. Yes.

Q. And the Delta represents the extra amount of particulate matter in the air in North Carolina alleged to result from what the plaintiff calls TVA's excess emissions?

A. Yes. And what I was speaking of was the Delta between what TVA projects would be its impacts on North Carolina in 2013, and the difference between that level and what would accrue if the Clean Smokestack caps were adopted.

MR. LANCASTER: Your Honor, may I approach the easel?

THE COURT: Yes.

BY MR. LANCASTER:

Q. Is Defendant's Exhibit 364 a summary you prepared to illustrate what we call the TVA Delta, the extra emissions, assuming the Court were to accept TVA's projections instead of Dr. Staudt's projections?

A. Yes, it is. And perhaps I should explain a little bit about what this display is.

In my report, I have many of these displays for almost

all the outputs I received, 2002, 2013. What this one displays is, across the bottom, is counties in North Carolina ranging from the western counties to the eastern counties.

And Your Honor, you will find this is Figure 36 in my first report. I don't know the page number.

MR. LANCASTER: I believe it's marked as Defendant's Exhibit 364 as well, Your Honor.

THE WITNESS: Your Honor, in my first report, that's dated February in '07, it is Figure 36, near the back of that report.

THE COURT: All right. Let me see if I can find that.

THE WITNESS: The title is *Annual Average PM2.5 in North Carolina Counties and Effective Additional Controls on TVA Delta for the year 2013*.

THE COURT: All right.

BY MR. LANCASTER:

Q. Dr. Anderson, if you could describe what this figure shows.

A. Yes. What it shows is that, on the horizontal axis, the counties in North Carolina are arranged from western counties to eastern counties as we go from left to right, and on the vertical axis are the concentrations of PM2.5.

The red line across the top, 15 micrograms per cubic meter, is the current National Ambient Air Quality Criteria

Standard for particulate matter on an average annual basis. And you will see them, there are bars created for each county that represents the total projected PM levels for counties across North Carolina in the year 2013, and the small red tip of each of those bars is that incremental, what's termed the TVA Delta. It's that incremental reduction that would be achieved should TVA adopt more controls, the Clean Smokestacks caps, I suppose it's called. So that's what those small red tips represent. They are obviously exceedingly small.

Q. Okay. And this is the TVA Delta. There's a separate North Carolina Delta that Dr. Levy used to make his calculations, correct?

A. That's correct. And I have reviewed the data, the modeling outcomes from Chinkin and Wheeler. They're similar, but in their modeled projections, they actually modeled lower projections for North Carolina across the board. So their bars are not quite as high.

And these small increments that I have termed the TVA Delta are -- the maximum is -- I think it's .06 on the 12-meter grid and .03 is an average. The Deltas that Spengler and Levy's -- Dr. Spengler and Levy's reports used, I think the maximum in any county was approximately .3, and the average across those counties was .15 micrograms per cubic meter. So they're slightly larger, but still very --

still very, very small.

Q. Well, I'd like to come back to the TVA Delta in a few minutes, but first I want to examine the implications of Dr. Staudt being right, the implications of the health impacts in North Carolina, of the North Carolina Delta, that assumes that TVA will have a much higher level of emissions in the year 2013 than TVA projects.

Are you aware that based on that information, Dr. Levy has prepared a calculation that the additional fine particulate matter in the air in North Carolina will cause 98 premature mortalities per year?

A. Yes, I am.

Q. And do you agree with that conclusion?

A. No, for a variety of reasons.

Q. Could you summarize those and then we can go through them in detail?

A. Yes. Maybe the best thing to do is place them in four categories.

The first category I will call modeling fatalities or mortalities at incrementally vanishingly small levels that are very much lower than where EPA has set the National Ambient Air Quality Criteria Standard to be protective of public health of sensitive individuals, to protect against mortality and morbidity, of 15 micrograms per cubic meter.

The second is the fact that the 98 fatalities for PM are

associated with very small Delta. We're not talking about large exposures. We're talking about very small amounts, .15 on the average, .3 micrograms per cubic meter maximum.

The third is the differential toxicity. There is no question and everyone agrees that when EPA decided to regulate particulate matter, to lump everything together in essentially a category of particulates, there was uniform recognition that this category contained different components. So it's a complex mixture. And depending upon its composition at any one location or point in time, the toxicity is going to be impacted. So I think the fact that we are speaking here of sulfates predominantly is an important factor.

And the final factor in their calculation is to calculate with such certainty a number of 98 fatalities for these incremental small amounts.

So I think those would be my four categories that caused me great concern about their calculations.

Q. Let's start with the first of those, the National Ambient Air Quality Standards.

During your work at the EPA, were you involved in any way in any development for setting the National Ambient Air Quality Standards.

A. Yes, I was. As I mentioned earlier, one year there, as the Deputy Assistant Administrator for the Air Program, as a

scientist, focused specifically on this program, and it was because this program is unique across all of EPA's landscape of legislative authorities, this particular one tells EPA that it must regulate to protect public health while considering nothing else. It is to consider only the science. So no feasibility to control, no cost to control, no benefit to control. It's to protect public health in the most sensitive individual from mortality, morbidity, and not only protect but with an adequate margin of safety, to be precautionary.

We recognized that this was a very difficult job because it's rare that science can define that very specific point on life. Science can put bounds around what's known and not known, but it's very difficult to define that one point. So I spent a year concentrating specifically on the implications of this very unique part of EPA's authorities and the scientific implications.

Subsequently, when I organized EPA's Central Risk Assessment offices, I organized and I was responsible for the -- my office was responsible for the group that wrote the National Ambient Air Quality Criteria. These documents are the underpinnings, the scientific underpinnings of the National Ambient Air Quality Criteria Standards.

Q. How does EPA go about selecting an ambient air quality standard?

A. It's a very long and laborious process. The process begins with the drafting of the Ambient Air Quality Criteria document. The staff often has outside experts help with different chapters of the document. There's an enormous body of literature that must be assembled and reviewed.

Usually, there are multiple drafts of the document. I think in this last work, on the particulate matter document, I believe there were five different drafts. Each draft of the document goes through peer review. It goes to the legislatively required body, the Clear Air Science Advisory Committee, the CASAC committee, as the acronym is called, for review. Public reviews, outside scientist review. There's the opportunity for the input of opinion from all the different groups of scientists, and then the criteria document is made final. It is kind of that risk assessment part of what I described earlier.

It then becomes the scientific basis for the regulatory office, the air office, to make decisions about what I call risk management, how to set the standard. And the -- that office, in the past, has written what is called the staff paper, which is to lay out the different options; considering the science, what would be the option for regulatory action. And the criteria document, the staff papers, are discussed, reviewed, made public, and commented on numerous times.

This last process I believe took a total of seven years,

and it's a very -- it's intended to be a very thorough process to incorporate all scientific opinion.

And then, finally, there's a final rule that's published in the federal registry that is EPA's decision. The Clean Air Act calls for five-year revisions. The agency has had difficulty sticking to a five-year schedule in many cases. But the agency is supposed to revisit these criteria every five years.

Q. And EPA, in fact, just set a PM2.5 ambient air quality standard that was finalized in late 2006; is that correct?

A. That's correct.

Q. And is EPA approach to setting a standard, like the particulate matter standard, is it a precautionary approach?

A. Yes. Well, the Clean Air Act anticipates EPA's responsibilities to be precautionary. That language is instructive to EPA. And in all of EPA's regulations, when we began or commenced the risk assessment approaches at EPA meaning the agency would accept risk, meaning not that the concept of accepting risk is we're accepting real impacts, but scientific uncertainties prevail. The agency has pursued a policy across all of its regulatory programs of being precautionary, meaning to be public health protective when it sets standards.

Q. And does EPA take into account sensitive sub-populations in setting standards?

A. I'm sorry, I didn't --

Q. Sensitive sub-populations. Does EPA take that into account?

A. Yes. And in the Clean Air Act requirements of EPA, EPA is supposed to be protective of sensitive sub-populations.

Q. And in your field, is the existence of an air quality standard, such as the recently adopted EPA standard for fine particulate matter, is that relevant to examining the public health implications associated with exposure to the pollutant?

A. I'm sorry. I'm not quite --

Q. I'm sorry. Is the existence of a standard, like the National Ambient Air Quality Standard, is that a relevant consideration in your field for examining the public health implications associated with exposure to a pollutant?

A. Yes, it is. It essentially is the culmination of EPA's long and arduous risk assessment process. And when they set the standard and specifically state that this is to be protective of public health of sensitive individuals and to protect against mortality and morbidity, it is -- it is a -- one of the EPA's most thoughtful processes and one of their most considered processes to arrive at a standard.

Q. And in this particular case, the facts from all of the computer modeling that each side has done projects that the entire state of North Carolina air quality levels will be

superior to what is required by the PM2.5 air quality standard.

A. That's correct. In the year 2013, EPA has also modeled what it projects across the country. EPA, the North Carolina modelers and TVA's modelers, project that North Carolina will be in compliance, that is, all the counties will be under, as we see here, under the National Ambient Air Quality Criteria Standard of 15 for particulate matter.

Q. And do you consider that standard to be effectively a threshold for risk analysis?

A. The standard, however we look at it, is effectively a threshold, because EPA has two ways of arriving at its standards. One is for carcinogens, where it assumes a linear non-threshold model. And, of course, PM has not been so classified. But EPA, even for carcinogens, where it emits, or if we don't have mechanisms of action but there is a possibility that there could be risk in a linear, non-threshold way, has accepted risk and has an acceptable risk range, so it regards risk lower than a tenth of minus six as de minimus.

For threshold agents, EPA has set levels routinely for threshold agents by ingestion, by referenced doses, and for inhalation, reference concentrations. This level is effectively, however we look at it, EPA arriving at a level they feel is amply protective of public health for mortality

and morbidity with an ample margin of safety for sensitive individuals and sub-population groups.

Q. But that is not inconsistent with the standard failing to be a zero risk standard, is it?

A. No. First of all, let's understand what we mean when EPA says it's not a zero risk standard. We decided in the early '70s that we couldn't achieve zero risk. It was very easy to understand why. When we pump gasoline, there is some level of risk that we know is associated with benzene emissions. Our drinking water standards accept some risk. And what this means is that it's not that we're accepting, in reality, that there are injuries occurring at these levels. Given scientific uncertainty, it's not unique with PM that most often there is no evidence for or against establishing exactly where harm commences. So EPA routinely, in every standard, virtually every standard it sets, unless we have some exceptional circumstances where we can define the mechanisms of action, will always be accepting the possibility that there is some risk below the standard, but without the knowledge scientifically that there really is any real impact. So EPA seeks to be protective in a precautionary way. So that's what EPA means when it says low risk.

In the early '70s, we actually tried to achieve zero risk and we found that to ban every risk possibility is not

an achievable goal, either socially or economically, in this country.

Q. You mentioned something called the Clean Air Scientific Advisory Council, CASAC for short, and so did Dr. Levy.

A. Yes.

Q. His testimony was that the CASAC advised EPA to set the standard, not at 15, but at a lower level, such as 13 or 14. Does that fact have any bearing on your opinion in this case?

A. No. No, it doesn't. And let me just go back to something I said earlier when I explained that this is a unique provision in all of EPA's legal authorities, that science is supposed to be the basis for selecting that one point. There can be debates about where that point is because science cannot always be definitive. The Clean Air Act Science Advisory Committee suggested to the agency a slightly lower level of 13 and 14. That's not that far from 15, but that's what they suggested. The administrator, in the end -- when I say administrator, it means the administrator signs these rules, but it's the agency and its deliberation considering all the scientific input -- said that the science -- the CASAC committee does not provide a scientific basis for choosing the 13 or 14, so the agency chose the 15 as being the most scientifically defensible point on that continuum.

But the other reason that it's not important to the

situation we face here is because even had EPA set a level of 14, North Carolina total particulate concentrations across the counties are projected for all of these counties to be below even that level. So had EPA set a slightly lower level, largely speaking, all of the counties across North Carolina are projected to be below even the level of 13. And I think using North Carolina's modeling, it's even a much lower, quite a distance below the level of 13.

Q. In other words, North Carolina's modeling predicts its entire state to be below the 13 level, not merely below the 15 level?

A. That's correct.

Q. You also mentioned that the extra exposure levels, the Delta attributable to TVA, is quite small. What bearing does that have on your opinion?

A. Well, when we are studying agents in general, and if the exposures are very high or we're talking about large amounts of exposure, obviously, we're quite concerned about health impacts here. I mentioned earlier that we're talking about very small amounts. The incremental amounts that Dr. Spengler and Levy reported in their document are the maximum 0.3 micrograms per cubic meter, and the average that we calculated from that data across the counties is 0.15 micrograms per cubic meter. These are very, very small levels. And then the TVA, what we spoke of earlier, those

levels are even smaller. The maximum is .06 micrograms per cubic meter from the 12-kilometer grid and .08 from the larger grid, the 36-kilometer grid. And the average is .03.

So we're talking about very, very, very small levels, and it's hard sometimes to grasp, when levels are small, what that really means. But when I thought about how small these levels are -- and we're not talking about something in a range where we have information that these levels cause any kind of impacts -- I started thinking of a way to frame what these levels really mean. Now, they are less than 1 percent of the National Ambient Air Quality Criteria Standard where EPA said it doesn't suspect any effects. They are very far below the chamber study that was reported in one of my papers, in the paper I co-authored, a paper by Schlesinger and Casey that reviewed the literature and reported that they didn't observe effects in human chamber studies until they got to a level of 1,000 micrograms per cubic meter on normal adults and 70 to 100 micrograms per cubic meter in asthmatics.

So we're speaking of human chamber studies where humans are exposed, granted, for usually an hour or two. But these would not even be ethical studies if we were seeing mortalities at levels of .15 or .3 of something as high as this in these chamber studies.

Also, I realize, and I wrote about this in my report,

that there is an asthma drug that is used routinely. It says it's safe for long-term use, that it allows that -- it's a sulfate suspension and it's -- I looked at the concentrations, and the daily dose that's prescribed on average is about 2 micrograms per cubic meter. So this means that someone being exposed, as an asthmatic, is exposed to about 2 micrograms, as opposed to these very low levels. So we can frame how low these levels are by looking at other concentrations.

And then something else we can do is we can ask ourselves how logical it is, if we look at these very small levels, to think that 98 deaths are associated with this incrementally very small level of .3 maximum and .15 on average. What does that mean? Well, it means that if we believe these numbers are real, we would be projecting approximately 10,000 deaths at EPA's 15 micrograms per cubic meter, the Ambient Air Quality Criteria Standard. And does that make sense when EPA sets it to be protective against mortality and morbidity? The asthma drug, if we look at what people are prescribed to take, we would have about 120 deaths in North Carolina's population of asthmatics. So we can ask ourselves if projecting these deaths from these very small incremental amounts really make sense.

And so this is of concern because, to me, these incrementally small amounts to be associated with these

fatalities does not make common sense, and these levels are so far below where we've had human exposures in chambers where the Ambient Air Quality Criteria Standard has been set, that it's very difficult to frame them otherwise and to say they're vanishing and small.

Q. Thank you. And the third point that I understood you to mention had to do with a particular kind of particulate matter we have at issue here, sulfate; is that right?

A. That's right.

Q. And Dr. Levy testified that the particular component of the particulate matter attributable to TVA here is primarily sulfate. Are you aware of that?

A. Yes, I am. And I would agree with him, that that is typically the case with power plant emissions, coal-burning power plants.

Q. In other words, particulate matter -- particulate matter is actually a soup composed of many different kinds of particles?

A. That's right.

Q. And sulfate is only one of them?

A. Correct.

Q. And in your opinion, is there a basis to conclude that exposures to sulfate causes mortality at the levels of TVA's alleged contribution here, which is no greater than about 0.31 micrograms per cubic meter on an annual average basis?

A. There is no evidence, scientific evidence, that would support that conclusion that I know of. And this results from having studied this issue. I'm a co-author on a paper that we published in 2007. We had an expert committee review the literature and look very carefully at the issue, and I know of no scientific evidence that would support the toxicity at these very, very small incremental amounts of sulfate.

Q. And if you will look at Defendant's Exhibit 367 in your book.

A. Yes.

Q. Is that a copy of the paper on which you were a co-author examining the health impacts of sulfate and nitrate particles?

A. That's correct.

Q. And what was your conclusion from surveying the literature?

A. This paper concludes that the epidemiology studies are equivocal, but the signals that are given by the epidemiology studies are that sulfate is less toxic, but that because sulfate is highly correlated, meaning it appears in concert with particulate matter, in epidemiology studies it's very difficult to take the two apart in order to separate the sulfate from the particulate matter.

In the animal studies, we can go to the literature and

study what has been demonstrated in an animal study and we see across the body of literature the fact that sulfate is not toxic, certainly not at these incrementally small levels.

And that's the question we are addressing here. We're not trying to address a question of at any level can an agent be toxic, because most any agent can be toxic even water, if one is exposed to enough. We're trying to address the issue of toxicity at very incrementally small levels.

Q. Dr. Peden, I believe it was, said that he relied on a study by a person named Huang, H-u-a-n-g, examining the toxicity of sulfate. Are you familiar with that study?

A. Yes, I am.

Q. And do you believe that it supports the opinions here?

A. Well, no. The Huang study is what's called a panel study, a chamber study, and there were 38 individuals here. I'm sorry. It's a cap study.

Q. Right.

A. And there were 38 individuals that he followed. But the cap -- the concentrated air pollutant particulates were divided into two categories, and one was -- for the chamber study was the mixture of sulfate, iron and selenium. So it was not a sulfate study. The individuals were exposed to much higher levels. I recall, I think Dr. Peden gave a very high level in his testimony. I recall levels certainly up to 10 micrograms per cubic meter that were exposure levels for

the individuals in the study. And the author reports at the end of the study that it's very important to do further research. The last sentence in this study reports that it's very important to do further research to understand the mechanisms that would apply to environmentally important levels, such as what we're talking about here.

So I don't regard the Huang study as being informative for a number of reasons. It's not a sulfate study. It's at higher doses by far than what we're talking about here, and even the authors acknowledge that.

Q. The fourth point you mentioned had to do with uncertainty. Are you aware that Dr. Levy testified that he provided no quantitative uncertainty bounds or sensitivity calculations for his calculations of premature mortality theory?

A. Yes, I am.

Q. And do you have an opinion about whether that's an appropriate method?

A. Well, it's totally -- it's not appropriate. We have any number of reasons to know that there are large uncertainties. We commence with uncertainties about the mixture that we are dealing with here, knowing that we are talking about primarily sulfate secondary particulate formation from TVA to North Carolina. So that's a large uncertainty. He's treated all particulate matters as if it's the same.

Certainly, the dose response function is highly uncertain. I think he's used a number of different functions in the past. So that's an area of great uncertainty.

The exposure is uncertain. That's not something he's done. He's taken it from the modelers. But what that incremental Delta amount is has its own uncertainty. So there are numerous uncertainties that affect these numbers, including the idea that we are working with a non-threshold concept, meaning that, in the absence of information either to show there is or is not a threshold, that Dr. Levy has projected 98 certain deaths in a region of very small exposure, and he's done it with great certainty, as if this is a very certain outcome, and I think it's -- it's unfortunate, because in all of EPA's guidance and all the risk assessment background that all of us have who work in this field, uncertainty is a very important part because we have to admit that the science is uncertain.

MR. LANCASTER: May I approach the flip chart once again, Your Honor?

THE COURT: Yes.

BY MR. LANCASTER:

Q. Did Dr. Levy, in failing to characterize the uncertainty, actually follow the Red Book methodology he outlined?

A. No. And I have been involved with this methodology for

a very long time, for over 30 years. It has an identification. EPA has now incorporated mechanisms of action and it has an identification step. We are clearly dealing here with a mixture and not just particulate matter. So it has an identification. Where we're addressing the weight of evidence that an agent is in fact capable of causing disease, there is no discussion of the uncertainty, considering particularly that we're dealing with a mixture known to be predominated by sulfates.

In the dose response, which I always put as the second step, we know that there are uncertainties in the data, and Dr. Levy, I think, has acknowledged that he's used different functions to address the dose response. In the exposure effect method, the projections are the basis for the incremental Delta he has used and less exposure. There is uncertainty in any of that exposure information created with Delta, and there is uncertainty in that function.

The risk characterization step is the step where all this information comes together, and there is a discussion of the weight of evidence that an agent is capable of causing disease, and then the quantitative outcome, with some discussion of what confidence you have. There is no discussion of any of that. There is just a certainty of 98 deaths associated with the incremental amounts, without further discussion. So the steps in the Red Book are not

steps that I believe he has acknowledged or followed.

Q. Now, the opinions you've been expressing have been based on the assumption of North Carolina's Delta based on Dr. Staudt's emission projections; is that correct?

A. I'm sorry. I couldn't quite hear you.

Q. The opinions you've been expressing are based on assuming the validity of Dr. Staudt's projections, his high emissions projections, that lead to the North Carolina Delta; is that correct?

A. That's correct.

Q. And if the Delta is, in fact, even smaller than TVA's Delta, your opinion would be altered?

MR. GULICK: Objection. Leading.

THE WITNESS: Yes, of course it would, because then the exposure assessment part of the paradigm would be altered.

Q. Now, recognizing that North Carolina quantified its view of the alleged health impacts in such a way that premature mortalities associated with PM2.5 constituted some 98 percent of the claimed impact, you still examined the other health impacts alleged by North Carolina, didn't you?

A. Yes, I did.

Q. And what did you conclude?

A. I concluded very much the same, that the other health impacts that were projected are below the National Ambient

Air Quality Standards and that the incremental amounts are very small in association, and I discussed those observations in my report.

Q. In conclusion, is it your opinion that TVA's small contribution to PM2.5 and ozone levels in North Carolina will not pose a threat to the health of the citizens of North Carolina?

A. I believe they will not pose a threat. There is no scientific evidence to support the fact that such small incremental amounts are associated with a mortality or morbidity.

MR. LANCASTER: Your Honor, at this time I move to introduce Defendant's Exhibits 364 and 367 identified during the course of Dr. Anderson's testimony.

THE COURT: Let those be admitted.

(Defendant's Exhibit No. 364 and 367 received in evidence.)

MR. LANCASTER: And we have no further questions.

THE COURT: All right. Mr. Gulick?

CROSS EXAMINATION

BY MR. GULICK:

Q. Good afternoon, Dr. Anderson. You received your modeling results of your modeling inputs in this case from Dr. Tesche and Mr. Mueller, did you not?

A. Yes, we did.

Q. And you also received -- that was based, was it not, on TVA's Clean Air Plan that was provided to you by Mr. Mike Scott? Is that right?

A. I'm sorry. I didn't hear the question.

Q. Pardon me. Dr. Tesche and Mr. Mueller's modeling was based upon TVA's Clean Air Plan; is that right?

A. That's my understanding.

Q. And Mr. Michael Scott actually provided you his explanation of what TVA's plan provided; is that right?

A. I did not review in detail what TVA was planning. I had used the modeling outputs and I understood that Mr. Scott would be addressing these plans, but I did not review them in detail.

Q. But you did discuss them with Mr. Scott, did you not?

A. Sorry?

Q. Did you not discuss those plans with Mr. Scott?

A. I wanted to be assured that the modeling assumptions were supported, and I was assured by -- is it Dr. Scott? Mr. Scott? I was assured by Mr. Scott that that was in fact the case.

Q. And are you aware that Mr. Scott's plan or the plan of TVA was based upon -- its main assumption was the Clean Air Interstate Rule and the Clean Air Mercury Rule?

A. I think that's a question for a different witness. I am not -- I think I'm not the correct person to be discussing

that.

Q. So you don't know the answer to that?

A. I don't know the answer.

Q. If, in fact, it was based upon the Clean Air Interstate Rule, you're aware that that rule has been vacated, are you not?

A. Yes, I am.

Q. You're aware that the Clean Air Mercury Rule has also been vacated?

A. I don't -- I just happened to hear about the first. I'm not an authority on this.

Q. Your projections of -- your Exhibit 364 is based upon the projections that were given to you by Dr. Tesche and Mr. Mueller, were they not?

A. That's correct.

Q. If the vacation of the CAIR rule were to change the inputs into Dr. Tesche and Mr. Mueller's modeling, or to undermine that, that could affect your predictions about what is going to be the actual level of fine air particulate in North Carolina and elsewhere. Is that not the case?

A. I have no idea how the vacating of that rule impacts the modeling. I think that's a question for -- as I said, unfortunately, I can't answer it. Whether it does anything to the modeling, changes the modeling, I assume that the other witnesses can address that better than I.

Q. My question to you is, if it did change the underlying assumptions of their modeling so that they were not correct, that would affect your projections in your Exhibit 364; is that correct?

A. Well, I think the point is I can't know, one, whether it changes assumptions; two, I don't know whether it makes assumptions go up or down. So I think the point is that I don't know. But if I'm given different modeling results, I will deal with different modeling results.

Q. In regard to the NAAQS, during the time that you were with EPA, did the administrator ever reject the advice of its scientific advisers?

A. I can't remember whether the administrator always agreed with the Science Advisory Council or not. I just -- I don't recall. It's an advisory body and it's made up of a few individuals. The administrator has a great deal more information to assimilate, including many, many outside scientific comments, and the criteria document is normally just an enormous document by internal scientists, and the administrator has to, in the end, consider the advice of the CASAC committee, but not follow it. They are not the dictators of the standard. So I can't tell whether there's ever been a discrepancy or not.

Q. So you can't -- you don't know the answer as to whether --

A. I don't know. I don't know. But I can tell you that there has never been a point in time when I was in the agency that the administrator felt compelled to be instructed to set a standard just exactly the way the CASAC committee suggested.

Q. The CASAC committee, with respect to the PM2.5 standard that was last set, which was a few years ago, recommended that it be lowered, did they not?

A. Yes. We've talked about that. They recommended a slightly lower level of 13 to 14.

Q. And they recommended that it be lowered because there was clear evidence, they indicated, that there were adverse health effects below the 15 micrograms per cubic meter standard. Is that not right?

A. I think that is not correct because, in the end, the administrator said that, while they suggested the slightly lower level, they did not provide a convincing scientific basis for that level. They just had a feeling that it should be slightly lower. And I discussed earlier that it really doesn't matter in this particular situation whether it's 13 or 15. It's not that much different. But the administrator decided that the scientific -- the solid scientific foundation supported the 15 and not the slightly lower 13 or 14.

Q. Is it not in fact the case that the chairman of the

CASAC wrote to the administrator on December 29, 2006, and stated, quote: The CASAC recommends changes in the annual fine particle standard because there is clear and convincing scientific evidence that significant adverse human health effects occur in response to short-term and chronic particulate matter exposures at and below the 15 micrograms per cubic meter, the level of the PM2.5 standard, unquote.

Isn't that the case?

A. Yes. I read the letter and I am aware of the letter, and I said that the administrator, after considering all the science, said that the committee said that but that they didn't provide a scientific basis. They didn't point to specific studies or cites that the administrator could use, and so the administrator decided it was amply safe to set the level at 15.

Q. Isn't it in fact the case that the administrator is a political entity?

A. I explained earlier, it's the administrator who signs all of EPA's authorities such as this. It doesn't mean that the administrator sits in his or her office and makes that decision. There is a great deal of scientific input. I was sitting there with my office when these decisions were being made for many years.

It is a highly deliberative process with a great deal of scientific input, so it's really unfair to give any taint to

this decision as a political decision.

Q. I'd like to show you --

MR. GULICK: Could you bring up the deposition, Gary?

BY MR. GULICK:

Q. I'd like to go to your deposition transcript at page 84, and it will come up on the screen. At lines 10 to 16. You see that in front of you?

A. Yes. Well, of course, the administrator is a politically-appointed --

Q. Well, let me ask you what I asked you.

Does the question not say: "No. I was asking whether the appointment is a political appointment"?

And is your answer not: "Yes, the appointment is a political appointment"?

Then you went on to explain. But is it not the case that you testified that the appointment is a political appointment?

A. There is no debate that the administrator of EPA is appointed by the president.

Q. Thank you.

In fact, in this case, did you not testify in your deposition that you actually did not do a risk assessment in this case?

A. No, I didn't, because EPA had done a risk assessment

over seven years, and I didn't feel that I needed to go back through that entire process. And that, in fact, is the risk assessment process.

Q. You mean setting the NAAQS? Is that what you're referring to?

A. Yes. In setting the National Ambient Air Quality Criteria Standard, EPA is basically reviewing all the risk information and arriving at a level that it feels is safe.

So what I did was review the exposure information, meaning the modeling outcomes, to essentially look at whether or not there would be risk either from the entire contributions to North Carolina from all sources or particularly from the incremental levels from TVA under different circumstances.

So I did conduct, in my view, an assessment, a risk assessment, but EPA had done the health-based assessment in a seven-year process, and I did not try to redo that.

Q. You did not try?

A. I said I did not try to redo what EPA had taken seven years to do. No, I did not try to redo that.

Q. EPA, in setting the NAAQS, was not considering the impacts of TVA's emissions on the state of North Carolina, was it?

A. Well, the NAAQS is anticipating nationwide impact, so, in that sense, it would embrace or would include or cast its

meaning over any contribution from any source.

Q. In this case, you did not consider the impacts of TVA's emissions on the state of Tennessee, did you?

A. Well, I did, in my report, include the entire domain, but as I said in my deposition, I thought the case was about impacts to North Carolina so my analysis primarily focused on North Carolina.

Q. So the answer is no?

A. I think I just gave the correct answer.

My report has plotted the whole domain. It shows TVA's contributions from the modeling at 1 percent, 2 percent and 5 percent in the whole domain, and those figures are presented in my report. But then I concentrated, by analysis, on North Carolina because it was my understanding that this case was about impacts to North Carolina.

Q. Like to show you what's been admitted as Plaintiff's Exhibit 148. It will come up on your screen.

This particular document has already been admitted into evidence. And I'd like to go to the second page of this document.

Are you aware, Dr. Anderson, that there are nonattainment areas for PM2.5 in the state of Tennessee?

A. I think I would need to go back to my maps. That's what I'm aware of. I have not seen these particular displays before. And I know in 2013 there are no projected

nonattainment areas either in Tennessee or North Carolina, as I recall.

Q. In doing your evaluation and in preparing your expert report in this case, you did not consider current impacts of TVA's facility -- of TVA's emissions on North Carolina or Tennessee. Isn't that the case?

A. Well, I did in the sense that, you know, Dr. Spengler and Levy did not, but I did -- I had modeling results for the year 2002, and then I had some additional information that told me that what was modeled for the year 2002 was reduced by 2005, I believe, by about 15 percent.

So I did consider all of the modeling information I had, and I considered the current contributions and I presented extensive plots like this, showing both the entire particulate contribution from the modeling in 2002 to North Carolina. I even broke out the contribution of TVA for the annual contribution to particulate matter, the 24-hour contribution, and also I did the same thing for ozone. And then, in addition, I displayed the monitoring results from North Carolina for both particulate matter and ozone and showed what counties are in nonattainment, and I testified in my deposition that those were 2002 data, and I believe there were three counties that were, from the North Carolina monitoring information, not in attainment. And then in the rolling average going forward, I looked up the information,

and only two of the three counties remained.

So, yes, I did consider it.

Q. Are you aware that the Environmental Protection Agency found that sources of SO2 and NOx in the state of Tennessee are contributing, or will contribute, significantly to nonattainment of the PM2.5 NAAQS in Catawba County and Davidson County in the year 2010?

A. I'm not aware. I don't know what you speak of.

Q. That's a technical finding in support of the CAIR rule.

A. I don't have that information.

Q. Did you know that --

A. But I do have --

Q. -- the U.S. -- let me ask you, did you know that the USEPA, as part of its support of the CAIR rule, found that sources of SO2 and NOx emissions in the state of Alabama contributed significantly to the nonattainment of the PM2.5 NAAQS in Catawba and Davidson Counties in North Carolina in 2010?

A. I cannot discuss the CAIR rule. I know it's been vacated. I did not discuss the document and I don't -- to be fair, I would have to know what incremental amounts we're speaking of. And what I do have are EPA's modeling results for the year 2013, 15 and 20, and I have North Carolina's modeling results for 2013 and the incremental contributions, and I have TVA's. I just don't have the -- I have not

reviewed this particular document.

Q. I'd like to draw your attention to -- I don't know what exhibit it is, but it's in your first expert report. And go to Figure 5-A. Have you been able to find it?

A. Yes.

Q. And the title of this figure is: "TVA Contributions to Annual Average PM2.5 Concentrations in North Carolina Counties for the year 2002." Is that correct?

A. That's correct.

Q. And this is based on modeling you received from Dr. Tesche?

A. Sorry?

Q. And this is based upon modeling that you received from Dr. Tesche and Mr. Mueller; is that correct?

A. That's correct.

Q. And is it not the case that this particular figure shows, by county in North Carolina, the contributions in 2002 as figured by Dr. Tesche and Mr. Mueller for -- the yellow shows the contributions from three plants in eastern Tennessee. Is that correct?

A. That's correct.

Q. And the blue shows the contributions from TVA's two plants in Alabama. Excuse me. In middle and western Tennessee. It has four plants; is that right?

A. That's right.

Q. I got my colors mixed up.

And the green shows the contributions to each county in North Carolina from TVA's two plants in Alabama?

A. That's correct.

Q. And the red, finally, shows the contributions from TVA's two plants in Kentucky to each county in North Carolina; is that right?

A. That's correct, yes.

Q. Now, these are annual average concentrations, are they not?

A. Yes. And the vertical axis displays those concentrations. It's .1, .2, .3, .4. So these are not the same. I didn't want you to think it was the same vertical axis as here. These are obviously much smaller contributions.

Q. I understand that. However, these are annual averages, and, in fact, they're going to be additive in the effect, are they not?

(Interrupted by court reporter.)

Q. Because these are annual averages, they're going to be additive to each county; is that correct?

A. Yes. We were breaking out and -- we displayed the total contribution from all of TVA and the counties as well, but we were breaking out each single contribution from the Kentucky, the Alabama and the western Tennessee and eastern Tennessee

plants because I was given the information; so that's what I displayed.

Q. I understand that. But, in fact, because they're annual average concentrations, they will be additive in each county?

A. If that's what the modeling by zeroing out means, then they would be additive. But we have the results additive as well displayed.

Q. So the answer is yes?

A. Well, I said I'm not a modeling expert. If that's what zeroing out means, I assume that, if we add them up, they will equal the diagram that I presented where they're all put together.

Q. So you don't know the answer?

A. I said I think that's correct, that if you add them all up and you look at the other diagram, you can put them together. I assume that you add them all up. They were zeroing out when they did the modeling. I assume that there is nothing in the model that prevents you from putting them back together to get the total contribution.

Q. Thank you.

With respect to Dr. Levy's estimates, he in fact described not his expert opinion but his best central estimate; is that correct?

A. That's what he describes it as. I don't know what his -- I didn't see anything else described, so I really

don't know what that means.

Q. But that was his description of it, was it not?

A. I believe that's what he said.

Q. And have you seen the -- have you reviewed the CAIR rule?

A. I just said I have seen it, but I have not spent time reviewing the CAIR rule.

Q. Were you in the court when I was discussing it -- when I was asking Dr. Moolgavkar about it?

A. Yes, I was. I -- and I have some familiarity about what was done there.

Q. In fact, is it not the case that the USEPA estimated that there would be 17,000 fewer premature deaths per year as a result of the controls of the cap-and-trade program they were imposing --

A. I saw that.

Q. -- in the United States?

A. I saw that that's what you were displaying, and I think, if I'm permitted to, I would like to add some perspective.

What EPA is obligated to do, particularly since OMB passed its rule that every time EPA sets a standard it has to present a cost-benefit analysis, EPA has been forced in more recent years to do this kind of work. And it's done because they have to do it, and they have to do it for OMB.

They do not use that kind of effort as a basis for

setting the Ambient Air Quality Criteria Standard, but, rather, they use that information to get a perspective for alternative -- kind of a sensitivity analysis for alternative decisions and then for their cost-benefit analysis and their submission to OMB.

Q. In fact, it was used to support a rule in which it's going to require installation of pollution controls on power plants in the United States. Is that not right?

A. I'm sorry. It was used how?

MR. GULICK: Madam Court Reporter, did you get my question?

COURT REPORTER: Yes, I did.

MR. GULICK: Could you read it back?

(The pending question was read by the reporter.)

THE WITNESS: I don't know if that's right, because when you say "support," I would need to know what you mean by support, and I haven't reviewed that information, so I don't know what you mean.

Q. You just said that it was a decision-making tool, did you not?

A. No, that's not what I said.

I said that until OMB got involved with the regulatory impact assessment requirements, and in my years at EPA, we were not trying to calculate impacts in this fashion.

EPA looks at potential impacts of particular rules, of

particular judgments, and it became an approach for doing that because it's required to do so in its regulatory impact analysis. Even though the Clean Air Act says when it sets the National Ambient Air Quality Criteria Standard, it must do it based only on the science and protection of public health when it looks at the impacts of what rules it's setting, then it makes -- it does this kind of impact analysis, and it's been -- it's been problematical for the situations like this where the science is very uncertain.

In fact, the agency went to the National Academy of Sciences to try to get help on how they could go about doing this.

Q. You heard Dr. Moolgavkar's testimony about the articles that he authored with you, starting in the middle of the 1990s, on the subject of air pollution?

A. Yes.

Q. And, in fact, is it not the case that the American Iron and Steel Institute was a client of your company?

A. I think it's -- yes, and that's true, and I would like to add a perspective to that as well.

When I was at EPA, the American Iron and Steel Institute regularly spent all of its money in legal contests with the agency. When I left the agency, I spoke with them. They asked me my opinion about the Ambient Air Quality Criteria Standards, and they said the agency would not listen to them.

And I said they're not going to listen to you on a legal basis, but if you are willing to sponsor some good scientific work, the agency will listen and they'll be obligated to listen. And so they did sponsor scientific work, and this was some of the work they began sponsoring. And I thought it was a good step. I think private sectors should be responsible for some of the investigations.

Q. At your deposition I asked you if you were familiar with an article called *Short-Term Effects of Air Pollution on Heart Rate Variability in Senior Adults in Steubenville, Ohio.* Do you recall that?

A. Yes, I do.

Q. Did you subsequently -- you had not seen that article, as I recall?

A. Yes. Of course, I looked it up. I have not seen it. It was published in 2008. You asked me about two articles, I thought.

Q. I think it was published in 2006. Did you say 2008 or did I just --

A. I thought you were talking about the Steubenville -- which article -- maybe we should resolve what we're speaking of.

MR. GULICK: I, unfortunately, only have one article, Your Honor, so I could put it up on -- I can show it to her and then put it up on this.

THE COURT: That will be fine.

MR. GULICK: We can put it up on the screen.

THE WITNESS: Could I have a copy of the article?

MR. GULICK: I'm going to display it to you.

THE WITNESS: I want to look at the whole article, not just selected excerpts.

MR. GULICK: Well, let me do this. Let me show you the discussion part, and before I ask any questions, I'll give the article back to you. How about that?

THE WITNESS: All right.

MR. GULICK: Is that fair enough?

THE WITNESS: That's fair enough.

BY MR. GULICK:

Q. First of all, this was one of the articles that we had -- that we had discussed during your -- or that I had shown you during your deposition; is that correct?

A. Yes. I think you showed me two articles. This one and one by Sarnat in Steubenville.

Q. But this was one of them?

A. I think so, yes.

Q. And what I want to draw your attention to is this discussion, and if you could focus in on the -- do I have to focus this in?

I'm sorry. I'm a complete amateur at this.

Have you read the first paragraph?

A. Yes.

Q. I'm going to move it down so that -- before I give it back to you --

A. All right. Thank you.

Q. -- so that we can read the second paragraph.

A. All right. I've read it.

Q. Is it not in fact the case, Dr. Anderson, that this study suggests that there is an association -- indeed, an association -- between sulfate exposure and changes in heart rate variability.

A. This is what is called a panel subject. It has 32 subjects. And what they were monitoring is heart rate variability.

First of all, to take one study when there are many studies, many panel studies that show some positive, some negative, is not -- to look at the study alone is not that informative, but looking at the study alone and the particular issues here, which is why I read it after the deposition, we're speaking of North Carolina where we're speaking predominantly of sulfate emissions. This study is in an area, Steubenville, Ohio, that's highly industrialized, where the pollution levels, the concentrations are much higher than here in North Carolina. There are many industries there that are not here in North Carolina.

The correlations here that were made with particulate

matter and sulfates did not discuss other potential contributions of metals and other types of particulate matter that could be involved, so I didn't think this was particularly useful to the discussions we're having here, both because it's one of many panel studies that show many different results and also because Steubenville is very different -- Steubenville, Ohio, is a very different profile than here.

There's also a second paper in Steubenville by Sarnat that looks -- in a similar panel study, and he, in his follow-up paper, references our paper, saying that -- and this was in 2008 -- that the results on effects of sulfate is far from resolved, and he references the paper that I co-authored.

And the other problem with this paper is heart rate variability is well known to be, as a background matter, highly variable in elderly people. So to try to superimpose heart rate variability on the effects and correlate them with effects of any particular exposure is a very difficult matter. And on the expert panel, that was that panel for the paper that I co-authored, Dr. Venditti, who is a leading cardiologist, questioned quite openly -- and he's written a paper on this -- that heart rate variability is not really a very good measure for detecting effects of pollutants.

Q. Well, I'm going to --

A. So, I mean, I did read this, and that's the context.

Q. You're aware of Dr. Peden's testimony that reduction in heart rate variability is a well known precursor for arrhythmia and more serious cardiovascular events?

A. I would say his statement is at odds with Dr. Venditti, who said that heart rate variability, first of all, if it's a 5 to 10 percent difference, it doesn't matter, and if it's -- heart rate variability is usually symptomatic of something. If it's more than that, it shows an underlying problem and not an etiological agent.

But I'm not a cardiologist, so I can only report what I know. We have differing opinions here from one person who is a renowned cardiologist and one who is not really a cardiologist. So I would tend to put the weight on the other side, but I can't resolve the differences.

Q. In fact, that is a matter about which you cannot testify. Is that right?

A. I'm sorry?

Q. You're not a medical doctor?

A. No, I'm not a medical doctor.

Q. So you're just repeating what you say that that doctor said; is that right?

A. Well, you were asking me --

Q. I'm just asking you, you were just repeating what --

A. You were asking me what Dr. Peden said, and you were

holding that out as an authority statement, and I'm just saying there are many different sides of the issue, and I think I'm perfectly at liberty to point out that Dr. Peden -- that they are differing opinions. And I can point those out. I said I can't resolve them.

Q. And Dr. Peden has given sworn testimony in this case.

MR. GULICK: No further questions.

MR. LANCASTER: No redirect, Your Honor.

THE COURT: Dr. Anderson, that will complete your testimony and you are excused. Thank you.

MR. GULICK: Actually, Your Honor, I would like to put that particular article into evidence, with your permission, and will mark it and make copies for the Court and for --

THE COURT: And what number will you give that?

MR. GULICK: That will be 493, Your Honor. And that was the article we were just discussing.

THE COURT: All right. Let that be admitted.

(Plaintiff's Exhibit 493 received.)

MR. GULICK: I will bring copies to the Court as well as to other counsel.

THE COURT: That will be fine. Take a recess until 2:30.

(Recess.)

[END OF VOLUME 10A]

UNITED STATES DISTRICT COURT

WESTERN DISTRICT OF NORTH CAROLINA

CERTIFICATE OF REPORTER

I certify that the foregoing transcript is a true and correct transcript from the record of proceedings in the above-entitled matter.

Dated this 26th day of July, 2008.

S/ Karen H. Miller

Karen H. Miller, RMR-CRR
Official Court Reporter